CAFFEINE CONSUMPTION, EXPECTATIONS OF CAFFEINE-ENHANCED PERFORMANCE AND CAFFEINISM SYMPTOMS AMONG NEW ZEALAND ATHLETES

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" ... sport is the play of the spirit, the challenge of the mind,
and the perfection of the body - not a contest of pharmacology."
(Wadler & Hainline, 1989)

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ABSTRACT

During the 1988 Olympic Games, an Australian pentathlete was ousted when a random urine test proved positive to a prohibited amount of caffeine after competing in 12 hours of fencing. This thesis begins with a literature review about caffeine in general, and about its use in sport.

The present research was carried out to investigate the incidence of caffeine abuse among New Zealand athletes, and their use of caffeine as a performance enhancing drug. A positive association amongst expectancies, caffeine consumption and caffeinism signs was predicted. It was also hypothesised that athletes who use caffeine as a performance enhancing drug would identity more strongly with the athlete role.

A survey questionnaire was designed to collect self report data on: daily caffeine consumption, DSMIII-R caffeinism symptoms experienced after consuming caffeine, expectations of caffeine-enhanced performance, and the extent to which subjects identify with the athlete role.

Respondents were 185 athletes across twelve sporting codes. The data were analysed using multiple regression, chi-square analyses and analyses of variance. 24.3% of the athletes reported using caffeine as a performance enhancing drug. 24.9% endorsed more than 5 symptoms of caffeinism. 26.5% of the sample believed caffeine can enhance sports performance and 51.4% of the sample identified strongly with the athlete role.

This study found a significant relationship between expectancy scores and caffeinism symptoms at the .01 significance level, but no relationship was found between expectancy scores and caffeine consumption. A statistically significant relationship was found for athletic identity and use of caffeine as a performance enhancing drug ($p<.001$).

The results of this study support the suggestion that caffeine be included in drug education programmes despite its ubiquity and legality. Suggestions for future research are made and implications for athletes are discussed.

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GLOSSARY OF ABBREVIATIONS

5-HT	serotonin
ACTH	adrenocorticotropic hormone
Ca^{2+}	calcium ion
c-AMP	cyclic adenosine monophosphate
CHO	carbohydrate
CNS	central nervous system
CO_2	carbon dioxide
COMP	In-competition Testing
CRF	cortico-tropin releasing factor
EIB	exercise-induced bronchoconstriction
EPI	epinephrine
FADE	Foundation for Alcohol, Drug Education
FFA	free fatty acid(s)
GABA	gamma-aminobutyric acid
H^+	hydrogen ion
IOC	International Olympic Committee
$\mu g.ml^{-1}$	micrograms (of caffeine) per millilitre (of urine)
$mg.kg^{-1}$	milligrams (of caffeine) per kilogram (of body weight)
$mmol.l^{-1}$	millimols (of caffeine) per litre of blood
NE	norepinephrine
NSOs	national sport organisations
NZOCGA	New Zealand Olympic and Commonwealth Games Association
NZSDA	New Zealand Sports Drug Agency
OOC	Out-of-competition Testing
PEDs	performance enhancing drugs
RER	respiratory exchange ratio
RPE	rating of perceived exertion
SICW	Second International Caffeine Workshop
VO_2max	maximum oxygen uptake

PART ONE

LITERATURE REVIEW

Introduction

During the 1988 Games in Seoul, a pentathlete became the first Australian to be expelled from the Olympics over drugs. His urine test showed an abnormally high level of caffeine following twelve gruelling hours of fencing competition (Christchurch Press, 1988a&b). This disqualification implied that the caffeine augmented his performance. Athletes select caffeine-containing beverages for many reasons, probably in part for their physiological and psychological effects. The belief among athletes that caffeine is an ergogenic aid is common, and several governing bodies of sport have barred use of the drug during competition.

Chapter one of this paper provides a summary of caffeine research. The focus is first on the historical aspects of caffeine, its sources and properties. Then, literature discussing the pharmacological effects of caffeine on the various body systems is presented. Chapter two discusses doping controls and some of the methodological issues relating to the ergogenicity of caffeine in sport. Finally, some of the conflicting evidence as to caffeine's use during different types of exercise is presented.

Care has been taken to present the research in a scientific and unbiased manner. However, philosophical and psychological implications are necessary when discussing the emotionally packed issues involved in the use of performance enhancing drugs. Every study has limitations. No study can be completely controlled, contain no false assumptions, or cover every eventuality. However, coaches and athletes are seeking factual and scientific evidence concerning the use of drugs, and the following review will give that evidence for caffeine.

CHAPTER ONE: CAFFEINE

Historical Aspects

It is thought that Palaeolithic man first discovered caffeine in plants and used it in drinks (McNaughton & Minikin, 1990). Certainly its consumption goes back as early as the origins of tea in China around 4700 BC. The appeal of caffeine lies primarily in its central nervous system (CNS) stimulating effect eulogised in the literature. According to legend, an Arabian goat herder in Yemen (c.850AD), noted his goats frisking around after chewing the berries of coffee beans (Murray, 1988). At the Ancient Olympic Games and in Ancient Egypt athletes ingested various substances believed to improve their physical capabilities. Similarly, Roman gladiators and knights in medieval jousts used stimulants (perhaps caffeine) after sustaining injury to enable them to continue in combat (Waddington & Murphy, 1993). During the 19th century, athletes also reportedly experimented with caffeine (Wadler & Hainline, 1989).

The recent explosion of recreational drug abuse in the Western World (Lucking, 1985) has added a new dimension to the problem of drug abuse in sport. Where caffeine used to be taken predominantly as a 'recreational' drug, athletes now often rely on its pharmacological properties for performance enhancement. Its basic action is to increase alertness and offset fatigue by affecting the CNS.

Although the media coverage of caffeine has not been as extensive as drugs such as anabolic steroids, it has been acknowledged. At the Seoul Olympics, Steve Hegg, a United States' cyclist and Alexander Watson, an Australian pentathlete, were both disqualified after failing drug tests for caffeine. Dyment (1987) even suggests caffeine is replacing amphetamine as the psychomotor stimulant of choice for many athletes. Indeed, a combination of caffeine and ephedrine (more readily available than amphetamine) may mimic the actions of amphetamine, producing the same type of central stimulation and reduction of fatigue in athletes (Lombardo, 1986).

The use of caffeine as an ergogenic agent cannot be denied. However, because of its ubiquitous nature, many people use this substance without acknowledging it as a drug. The effect of caffeine is generally expressed in the following way: "a mild stimulant, helpful in temporarily relieving

minor fatigue and boredom with little risk of any harmful effects" (Graham, 1978). It is a substance consumed by people in almost all age groups. Many soft drinks contain caffeine so people start consuming it at an early age. The sports world is often said to be a microcosm of society (Taylor, 1990; Hemery, 1986) so it is not surprising that athletes are consuming caffeine.

To date, only limited New Zealand data on caffeine consumption are available (Russel & Wilson, 1991). However, an Australian study (Leonard et al., 1987) determined the average caffeine consumption for adults as 240mg per day. The New Zealand average may resemble this due to similarities in eating patterns. The New Zealand population derives 70-80% of its caffeine from tea (Phelps & Phelps, 1988). The LINZ survey (Appendix 1 & 2) reports that the beverages most frequently consumed by New Zealanders are, in order: tea (23 cups per week); coffee (19 cups per week); and water (15 glasses per week; Russel & Wilson, 1991).

Sources of Caffeine

The compounds responsible for the stimulating action of coffee, tea, and cocoa are methyl derivatives of xanthine (2,6-dihydroxypurine). They are purine-based compounds containing two condensed heterocyclic rings, and are naturally occurring chemicals found in the leaves, seeds, and fruit of more than 60 species of plants. Caffeine is found in tea (*Thea sinensis*), coffee (*Coffea arabica*), cola nuts (*Cola acuminata* and *Cola nitida*), and many other plants (Graham, 1978). Caffeine is readily extracted from its plant sources and is very soluble in boiling water. Although the caffeine of tea leaves exceeds that of coffee beans, much more dilution is usually noted in a cup of tea (Falls, 1968). Plant variety, geographic location, climate and cultural practices including fermentation, influence the content of caffeine in natural products (Graham, 1978).

If not in what people drink, caffeine is eaten in foods - baked goods, chocolate, lollies and many processed foods as a flavouring agent (Watson, 1988). It is also consumed in common medications, both prescription and nonprescription. According to the Food and Drug Administration's National Centre of Drugs and Biologics, more than 1000 over-the-counter drugs show caffeine as a listed ingredient (Lecos, 1984). Standard average caffeine contents for various items, are shown in Table 1.

Table 1
Caffeine Content of Some Common Items

Beverage	Volume	Caffeine Content (mg) Average	Range (mg)	Equiv. in urine within 2-3 Hrs
Coffee[a]	150ml			
Brewed, drip method		115	60-180	1.5µg/ml
Brewed, percolator		80	40-170	
Instant		65	30-120	
Decaffeinated, brewed		3	2- 5	.03-.04µg/ml
Decaffeinated, instant		2	1- 5	
Tea[a]	150ml			
Brewed		50	20-110	
Instant		30	25- 50	
Cocoa beverage	150ml	4	2- 20	
Chocolate milk beverage	250ml	5	2- 7	
Milk chocolate	30g	6	1- 15	
Dark chocolate	30g	20	5- 35	
Soft Drinks[b]	375ml			
Mountain Dew		54		
Coca-Cola		45.6		.68µg/ml
Diet Coke		45.6		.68µg/ml
Pepsi-Cola		38.4		
Diet Pepsi		36.0		.54µg/ml
Prescription Drugs				
Cafergot (antimigraine)		100mg/tablet		
Migril (antimigraine/anti-emetic)		100mg/tablet		
Ergodryl (antimigraine)		100mg/tablet		
Nonprescription Drugs (Pharmacy Only Medicines and Over the Counter Drugs)				
Weight-Control Aids				
Medislim Weight Control Tablets		100mg/tablet or capsule		
Trim Tabs		50mg/tablet		
Alertness Tablets/Sleep Inhibitors				
NoDoz Awakeners		100mg/tablet		1.5µg/ml
NoDoz Plus		100mg/tablet		
Alert Tab		100mg/tablet		
Analgesic/Pain Relief				
Anacin, Maximum Strength Anacin		32mg/tablet		.48µg/ml
Diuretics				
Cold/Allergy Remedies				
Codral Cold & Flu		30mg/capsule		
Endacol-C		30mg/tablet		
Drixine Cough Suppressant		15mg/5ml		
Medi-Tab Hay Fever Tablets		100mg/tablet		

Note: Taken from Heel, 1989; Lecos, 1984; Slavin & Joensen, 1985; Wadler & Hainline, 1989 (Similar items are given different values for caffeine content by various authors.)

[a] **With coffee and tea, caffeine content also depends on brewing time.**

[b] **About half the caffeine comes from kola beans and half is added (Graham, 1978).**

Chemistry of Caffeine

Caffeine is a methylated xanthine (Figure 1). Xanthine per se is a dioxypurine, and it is structurally related to uric acid. Caffeine is 1,3,7-trimethylxanthine. The methylation of position 1 accounts for the CNS stimulation, the methylation of position 3 is predominantly responsible for the diuretic effect, and the methylation of the 7 position correlates with cardiac stimulation (Wadler and Hainline, 1989).

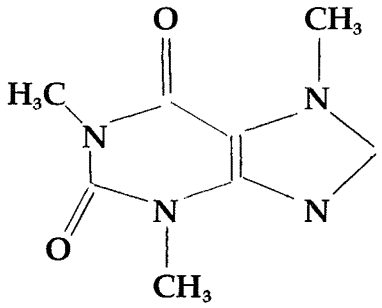


Figure 1 - The structural formula of caffeine, an alkaloid consisting two heterocyclic rings with three methyl groups attached.

Absorption, Distribution, and Metabolism

Caffeine empties rapidly from the stomach after oral, rectal or parenteral administration and is absorbed from the gastrointestinal tract (McNaughton & Minikin, 1990). With ingestion of 250mg, significant amounts appear in the plasma within 15 minutes (Powers & Dodd, 1985), and peak concentrations are reached at approximately 60 minutes regardless of dose (Bellet et al., 1968). However, there are subject differences in the distribution, metabolism and excretion of the drug (Sawyer et al., 1983).

Once absorbed, caffeine is distributed in approximate proportion to tissue water content. Thus dosage, body composition (Schreiber et al., 1988b), and hydration state influence a subject's physiological response(s).

There is no uniformity in the literature concerning the plasma half-life of caffeine (Curatolo & Robertson, 1983; Stavric, 1988) but the range appears to be between 3.0 and 7.5 hours. Factors influencing the rate of elimination of caffeine from the body include genetic factors, diet, urinary volume and pH, disease states, endocrine status, smoking, concurrent drug use such as oral contraceptives, pregnancy, and even ethnic origin (Davis, 1990; Delbeke & Debackere, 1984; Kuznicki & Turner, 1985; Sawyer et al., 1982).

Caffeine is primarily degraded via hepatic metabolism and its excretion as single methyl-group xanthines and methyl-uric acids is primarily renal, with little appearing in the faeces (Delbeke & Debackere, 1984). Approximately 3% of caffeine is excreted unchanged (Graham, 1978). The hepatic clearance of caffeine depends on hepatic blood flow. Caffeine's catabolism and elimination from the body during sports are decreased, relative to intensity of effort, due to sympathetic activity and the resultant decrease of blood flow to the liver (Duthel et al. 1991). Interestingly, van der Merwe et al. (1991) found that excessive sweating during endurance exercise does not enhance urinary caffeine concentration.

Mechanism of Xanthine Action

Xanthines share several common pharmacological actions, caffeine being the most active on the CNS. General actions include stimulation of: the CNS; the heart and skeletal muscles; the kidneys, inducing diuresis; respiratory rate and depth; and possibly the adrenal glands (Table 2). Xanthines may also induce relaxation of smooth muscle, stimulate gastric secretion, and elevate plasma-free fatty acid and glucose concentration. There is also the possibility that the drug may alter the release, binding, or activity of neurotransmitters in the brain, thus affecting 'perception' of work intensity. To provide the background information needed to appreciate caffeine's potential ergogenic effects, a discussion of caffeine's effects on the above tissues and processes will follow. Each disclosure will provide basic information concerning the qualitative pharmacological effects of caffeine.

Cellular Basis for Caffeine Action It appears that caffeine acts as a neurotransmitter. A number of cellular mechanisms have been proposed to explain the CNS stimulant effects of caffeine. Of particular importance are those actions of caffeine that might affect work performance by direct or indirect effects on the cardio-respiratory system, the nervous system, skeletal muscle and/or shifts in substrate metabolism. In order of caffeine sensitivity, these mechanisms are: translocation of intracellular calcium; actions mediated by the accumulation of cyclic-adenosine monophosphate (c-AMP); and actions caused by the blockade of adenosine receptors.

Table 2
The effects of caffeine on human physiology[a]

Physiological variable	Effects reported for caffeine
Respiration	
Respiratory rate	increase
O_2 Consumption	increase
CO_2 Elimination	increase
Cardiovascular effects	
Heart function	
Rate	increase, decrease[b]
Cardiac output	increase, decrease
Force of contractions	increase, decrease
Compensatory vagal activity	present
Circulation	
Coronary & Pulmonary	increase, decrease
Skeletal muscle	increase, decrease
Hepatic & Renal	increase, decrease
Cutaneous	increase, decrease
Cerebral	decrease
Peripheral resistance	increase, decrease
Blood Pressure	increase
CNS Stimulation	increase
Anxiety Reaction	
Anxiety, Restlessness	increase
Periods of depression	increase
Tremors, Flushing	increase
Diuresis, Insomnia	increase
Sensory disturbances	increase
Skeletal Muscle	
Neuromuscular transmission	facilitated
Power of contractions	increase
Fatigue	decrease

[a] **Adapted from Sawyer et al. (1982) and Passmore et al. (1987).**

[b] **Effects reported as increasing or decreasing are those where compensatory vagal activity plays a role in determining the final outcome.**

Firstly, *in vitro* studies of the effects of caffeine on translocation of intracellular calcium (Ca^{2+}) suggest that an alteration of Ca^{2+} permeability occurs in the sarcoplasmic reticulum of muscle. This results in an increase of Ca^{2+} release. This may augment the force of skeletal muscle contraction (Van Handel, 1983).

The second cellular mechanism of caffeine action is related to the inhibition of the enzyme phosphodiesterase (PDE; Waldeck, 1973). PDE catalyses the degradation of c-AMP to 5'AMP. Of interest in exercise is the role that c-AMP plays in regulating hormone-induced glycogenolysis and lipolysis (Wadler & Hainline, 1989).

Thirdly, through competitive antagonism, caffeine blocks adenosine receptors. The resulting blockade of the sedative effects of adenosine (Weinhold, 1991) leads to CNS stimulation (Powers & Dodd, 1985; Table 3).

Table 3.
The contrasting effects of adenosine and caffeine *

parameter	adenosine	caffeine (CA)
blood pressure	decrease	increase
urine output	decrease	increase
CNS activity	decrease	increase
lipolysis	decrease	increase
bronchial tone	increase	decrease
blood vessels	dilation	constriction
release of catecholamines	decrease	increase

***Adapted from Leonard et al 1987**
These effects are reversed in CA withdrawal and after the development of CA tolerance.

Effects on the Central Nervous System

Caffeine passes the blood brain barrier and is characterised as a powerful CNS stimulant affecting the medulla, all portions of the cortex and the spinal cord (Murray, 1988). It has many effects on the CNS and may influence psychomotor coordination, electroencephalographic (EEG) spectra, sleep, mood, behaviour and cognition (Curatolo & Robertson, 1983; Sawyer et al., 1981).

The molecular basis for caffeine's effects on the CNS is unclear. The behavioural effects could result from blockade of adenosine receptors, rather than from inhibition of PDE and the resultant accumulation of c-AMP (Jacobson & Edwards, 1990; Powers & Dodd, 1985; SICW, 1980). This is because the adenosine antagonist action occurs at much lower concentrations than are needed for inhibition of PDE (SICW, 1980).

Caffeine increases the catecholamine neurotransmitters, especially epinephrine (EPI; Graham & Spriet, 1991) and norepinephrine (NE; Bellet et al., 1968, 1969; Innes and Neckerson, 1975; Van Handel et al., 1979; Waldeck, 1973). This is consistent with a generalised stimulation of the sympathetic nervous system (Bellet et al., 1969) due to the direct effect of caffeine on the adrenal medulla. These observations suggest a possible conflict between the direct effects of caffeine on adenosine receptors and the indirect effects of caffeine-induced catecholamine release.

The arousing effects of stress, exercise and caffeine are similar (Lane & Williams, 1985; Raitliff-Crain et al., 1989). Caffeine stimulates the adrenal medulla to secrete hormones that affect the autonomic nervous system (cardiovascular and metabolic variables), and which are closely associated with emotion, stress and arousal (DeMeersman et al., 1986; Sawyer et al., 1982; Figure 2).

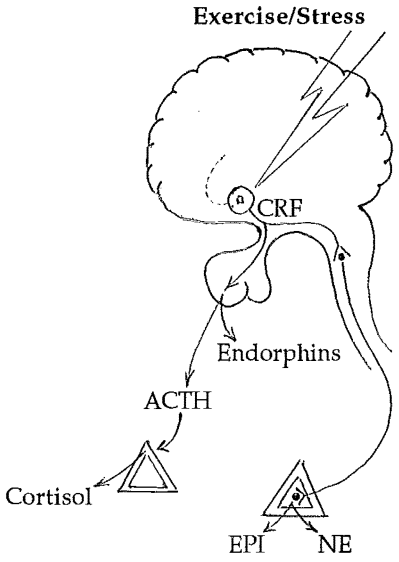


Figure 2 - Exercise-induced discharge of the corticotropin-releasing factor (CRF) with: (1) stimulation of ACTH and β-endorphin release from the anterior pituitary, followed by secretion of corticosteroids, and (2) activation of the sympathetic nervous system within the brain, followed by adreno-medullary secretion of EPI and NE. Caffeine potentiates this response. Adapted from Harber & Sutton (1984) and Sutton (1985).

Besides NE and EPI, caffeine elevates brain levels of serotonin (5-HT), and causes an increase, followed by a rapid decrease, in dopamine formation (Waldeck, 1973). Dopamine serves as a precursor for EPI and NE, and has its own receptors and functions: increasing heart rate, cardiac output, and blood pressure, as well as inhibiting insulin. Caffeine also affects gamma-aminobutyric acid (GABA) which contributes to the control of spinal reflexes, and acetylcholine release.

Evidence suggests that many of the cardiovascular, analgesic, and behavioural effects of exercise are mediated by endorphins, the brain's opioid (Farrel et al., 1982; Harber & Sutton, 1984; Sforzo, 1988; Sutton, 1985; Thoren et al., 1990). These effects could increase an athlete's ability to sustain physical effort (Shyu et al., 1982). Caffeine's effects on beta-endorphin secretion are not completely understood but it probably indirectly causes hypothalamic stimulation of pituitary β-endorphin secretion (Rodrigues et al., 1990). Caffeine also acts on hypothalamic releasing factors,

hindering the secretion of growth hormone and thyrotropic releasing factor. However, it seems unlikely that the doses of caffeine usually consumed would produce significant endocrine effects (Rodrigues et al., 1990).

Mood, Vigilance and Anxiety Caffeine is thought to have stimulant-like behavioural effects on mood (Kuznicki and Turner, 1986). The general belief is that caffeine allays drowsiness and fatigue, enhances perception of sensory stimuli, and reduces reaction time. However, conflicting data exist regarding caffeine's effects on concentration, drowsiness, fatigue and vigilance (Wadler & Hainline, 1989). Lieberman et al. (1987) and Kerr et al. (1991) observed significant effects of caffeine on vigilance but not mood state. Zwyghuizen-Doorenbos et al. (1990) found that caffeine (250mg) improved alertness and Goldstein et al. (1965) found increased alertness after 100-300mg caffeine. This facilitatory effect of caffeine on vigilance may be due to the drug combating fatigue-related decrements seen during control conditions (Goldstein et al., 1965). Caffeine's inhibition of PDE could account for this finding (Murray, 1988). Others (Lieberman et al., 1987) suggest that caffeine's positive effects are not limited to situations where fatigue is present, but may be more general.

Kuznicki and Turner (1986) suggest that an optimum caffeine dose might exist for peak alertness to tension ratios. Evidence indicates that high levels of caffeine consumption relate directly to high anxiety levels (Sawyer et al., 1982). Caffeine's influence on anxiety may interact with situational variables, such as the competitive sports arena, causing subclinical anxiety levels rise to rise to clinical levels (Davidson & Smith, 1991).

Caffeine-induced changes in anxiety, reaction time, vigilance, and motor skills may affect the efficiency, accuracy and stress levels of athletes. It is important to note that caffeine reacts with other drugs such as pseudoephedrine and phenylephrine. Also, caffeine reduces the performance impairments of the benzodiazepines (Johnson et al., 1990) but not alcohol (Smith et al., 1991). Regardless of the mechanism (direct or indirect), caffeine is able to act upon the CNS. It has marked effects on perception, alertness and wakefulness (Goldstein et al., 1965), and on fatigue during muscular work (Costill et al., 1978; Ivy et al., 1979).

Effects on the Heart and Vasculature

The cardiovascular effects of caffeine are mediated through the stimulation of the medullary respiratory centre of the CNS and/or direct action on the heart and vessels. All parts of the circulatory system are directly affected by caffeine and these actions may be antagonised by caffeine's stimulation of compensatory vagal centres in the medulla. For example, the cardiac muscle is strongly and directly stimulated by caffeine, increasing the heart rate and the force of its contractions. At the same time, caffeine stimulates the medullary vagal nuclei, which decreases the heart rate. This antagonistic action may result in bradycardia, tachycardia, or no change in heart rate (Murray, 1988). This might explain the various findings regarding caffeine's effects on heart rate. Some studies have reported an elevated exercise heart rate (McNaughton, 1986), others a slowing of heart rate (Horst et al., 1936) and others, no change in heart rate (Fisher et al., 1986).

Caffeine also has an antagonistic action on the blood circulation. It causes the smooth muscles of coronary, pulmonary, and general systemic blood vessel walls to dilate. It simultaneously stimulates the medullary vasomotor centre, which causes constriction of these vessels, decreasing cerebral blood flow. No compensatory activity by the medulla occurs there.

Smith et al. (1991) reported no effect of caffeine on pulse rate but a significant increase in blood pressure. Passmore et al. (1987) also reported increased diastolic and systolic pressures. The blood pressure response to caffeine depends on the balance between central vasomotor and myocardial stimulation, which tend to increase it (Van Handel et al., 1979) and vagal stimulation and peripheral blood vessel dilation, which tend to decrease it (Smith et al., 1991). This homeostatic mechanism results in few life-threatening side-effects from caffeine use. However, caffeine does alter the volume and distribution of blood, thus affecting substrate and waste product distribution during exercise.

The cardiac index increases following caffeine are probably due to enhanced contractility (Ivy et al., 1979). At an electrophysiological level, caffeine may affect the rate of calcium exchange, which is responsible for the heart's contractility. The increased release of calcium from the sarcoplasmic reticulum can result in an oscillatory potential at the beginning of diastoles, which could contribute to the onset of arrythmias.

The cardiovascular effects of caffeine differ between caffeine-tolerant and caffeine-naive individuals (Curatolo & Robertson, 1983). Whereas the latter may experience altered haemodynamics (pressor response and increase in heart rate), chronic caffeine ingestion has little effect on blood pressure, heart rate or plasma catecholamines (Myers, 1988).

Respiratory Effects

Caffeine appears to have a respiogenic effect, increasing respiration rate and oxygen consumption (Berry et al., 1991; Murray, 1988; Powers, 1983). The mechanism for these changes is unknown although as a respiratory centre stimulant, caffeine may: increase central chemosensitivity to carbon dioxide (CO_2) such that its elimination is increased (Powers et al., 1985); act directly to stimulate the respiratory medullary complex; affect the inputs from the respiratory chemoreceptors; and act directly on neuromuscular transmission of the respiratory muscles (Murray, 1988).

One of the pharmacological effects of caffeine is relaxation of the smooth muscles of the bronchi (Curatolo & Robertson, 1983). In 1990, Kivity et al. showed that as well as a general bronchodilating effect, caffeine also has a protective value on exercise-induced bronchoconstriction (EIB) in asthmatics. This effect is only seen at large doses ($7mg.kg^{-1}$) which seem impractical to ingest before exercise.

Effects on Skeletal Muscle

Caffeine distributes in the body water, and is therefore most concentrated in skeletal muscle (Power & Dodd, 1985). Because tissue response is proportional to xanthine concentration, significant effects on muscle could be expected and may include enhanced contractile status, altered patterns of fibre recruitment and substrate mobilisation, and shifts in the rate of substrate use. These actions are mediated by direct actions on muscle or indirectly through alterations in hormone status (Figure 3).

Contractile Status It is well established by *in vitro* studies that caffeine has a direct effect on muscle contraction (Hartree & Hill, 1924; Powers & Dodd, 1988; Waldeck, 1973). Evidence suggests that caffeine potentiates the contractile capacity of both fatigued and rested muscle stimulated *in situ*. (Bianchi, 1962; Huidobro & Amenabar, 1945) and facilitates neuromuscular impulse transmission (Huidobro & Amenabar, 1945; Waldeck, 1973).

Finally, Lopes et al. (1983) have demonstrated that caffeine has a positive effect on human skeletal muscle properties *in vivo* both before and after muscle fatigue. These phenomena are apparently related to alterations in the release, uptake, or storage capacity of the sarcoplasmic reticulum for calcium, a modulator of contractility.

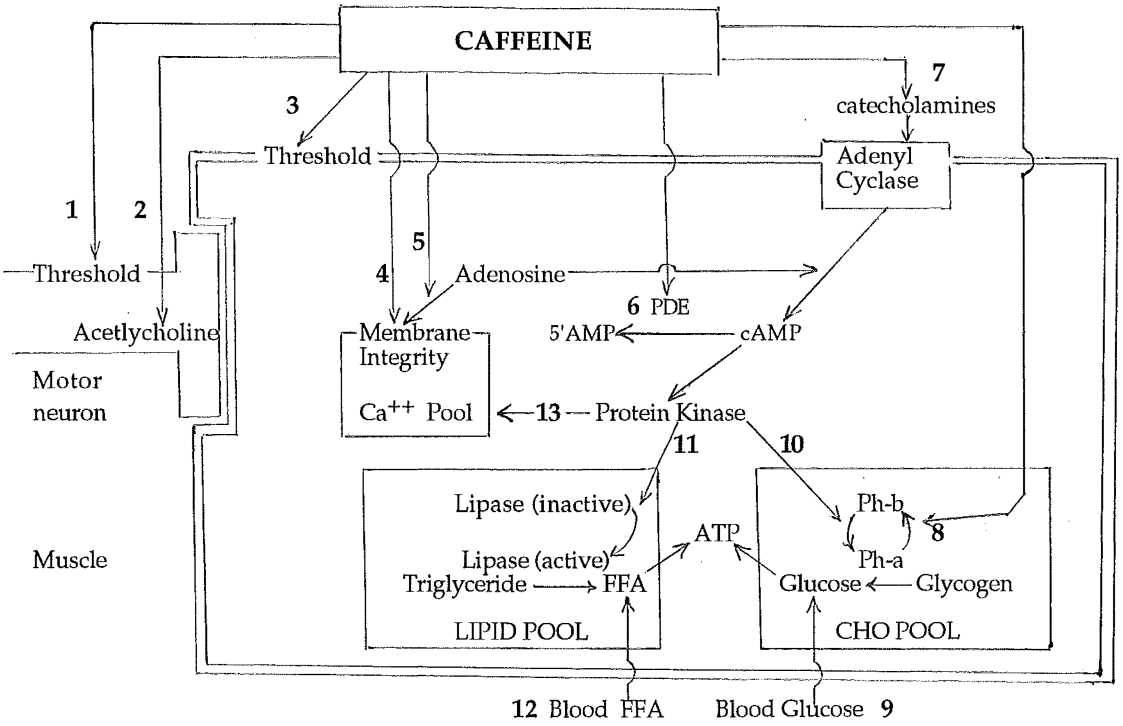


Figure 3 - Possible role of caffeine in the regulation of muscle actions. These include both direct and indirect mechanisms (clockwise from left): alteration of neuron threshold (1), stimulation of NT release (2), alteration of sarcolemma threshold (3), alteration of membrane activity related to calcium storage (4), inhibition of adenosine binding (5) and PDE activity (6), stimulation of catecholamine release (7), that, in turn, increases cAMP activity. The latter activates protein kinases that turn on glycogenolysis (10), lipolysis (11), and contractility (13). In addition, caffeine may inhibit glycogenolysis (8), increase blood glucose levels (9), and induce FFA mobilisation from adipose tissue (12). From Van Handel (1983).

There are four intracellular mechanisms by which caffeine directly exerts its effects on calcium. Caffeine may: rapidly release calcium from the sarcoplasmic reticulum; decrease the rate of calcium uptake by the sarcoplasmic reticulum; induce a loss of membrane integrity resulting in the increased calcium permeability of the sarcolemma; and, increase intracellular c-AMP levels by PDE inhibition. The last, in turn, may induce protein kinase-catalysed phosphorylation and increase Ca^{2+} uptake by the reticulum calcium pump. Caffeine's effects on skeletal muscle contractility may be mediated by all four of the above cited events. Williams (1991) reviews these mechanisms excellently.

Substrate Mobilisation and Use The second major area of caffeine effects on muscle is related to alterations in the lipid and carbohydrate storage pools (adipose and liver tissues, respectively) as well as direct actions on muscle itself (Spriet et al., 1992). Potentially these actions may provide the most dramatic effects on work output under *in vivo* conditions (Van Handel, 1983). The mechanism is dependent upon caffeine altering the concentration or activity of c-AMP or of several regulating enzymes.

Caffeine increases cellular c-AMP by two mechanisms (Figure 4). First, by sensitising central catecholamine receptors (Hartman & Becker, 1972), circulating catecholamine levels are raised (Bellet et al., 1968; Graham, 1978). This increases adenyl cyclase activity with consequently greater production of c-AMP. Second, caffeine inhibits PDE (Waldeck, 1973), the enzyme that catalyses the conversion of c-AMP to 5'-AMP (Powers & Dodd, 1985). The resulting increased c-AMP, in turn, gives rise to increased glycogenolysis (increased blood sugar) in caffeine-naive subjects and to increased lipolysis (increased free fatty acids) in all subjects (Curatolo & Robertson, 1983; Powers & Dodd, 1985).

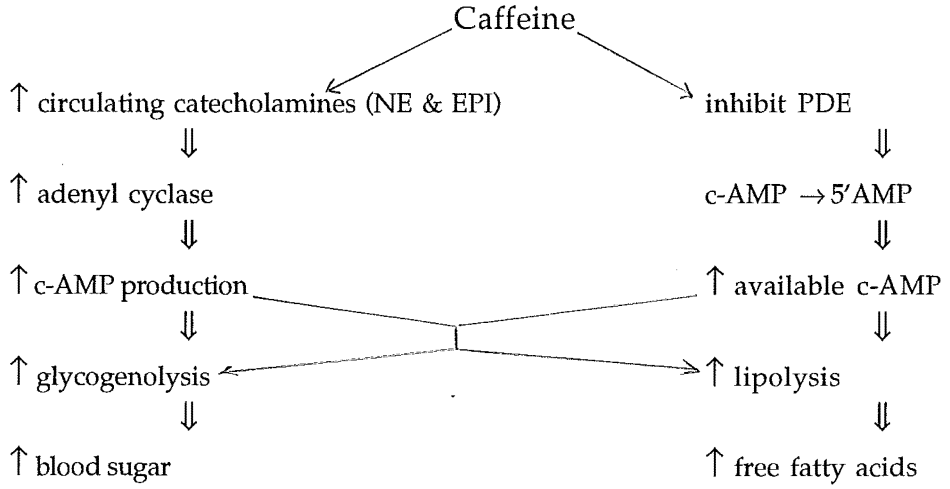


Figure 4 - Pathway of caffeine-induced increased glycogenolysis and lipolysis.

Glucose Homeostasis Caffeine alters glucose homeostasis by increased glycogenolysis, having a hyperglycaemic action (Cheraskin et al., 1967; Graham, 1978) through activation of phosphorylase in liver tissue. This is dependent upon either caffeine-induced increases in blood catecholamine levels (Bellet et al., 1969; Van Handel et al., 1983), or a direct inhibition of phophodiesterase (Waldeck, 1973; Figure 4). The resultant increase in c-AMP from either mechanism activates a protein kinase that accelerates phosphorylase activity, stimulating hepatic glycogenolysis (Bjorntorp, 1993). As a result of the stimulation of glycogenolysis, there appears to be a release

of glucose into the peripheral system that induces insulin secretion, potentially making the glucose available for peripheral uptake and metabolism. Whether this sequence of events occurs *in vivo* is unclear, however, since the adipokinetic effects (see below) of caffeine (Bellet et al., 1968; Weir et al., 1987) are well established and would likely suppress peripheral glucose uptake and oxidation.

Blood Lipids Caffeine stimulates lipolysis (Bellet et al., 1968, 1969; Costill et al., 1978; Essig et al., 1980, Ivy et al., 1979, McNaughton, 1986; Wilcox, 1982) in adipose tissue which results in increased plasma levels of free fatty acids (FFA; Costill et al., 1978; Essig et al., 1980; Powers et al., 1983). When ingested prior to exercise, caffeine has been reported to delay the onset of exhaustion (Costill et al., 1978: Ivy et al., 1979), due to the stimulation of FFA mobilisation (Bellet et al., 1965), leading to a glycogen-sparing effect (Essig et al., 1980; Spriet et al., 1992). The enhanced lipolysis is a result of the activation of lipase. Lipase activity can be enhanced by caffeine by the same mechanisms mentioned above (Figure 4).

First, caffeine causes an increase in blood catecholamine levels leading to increased intracellular c-AMP. This activates a protein kinase that accelerates the phosphorylation of inactive triglyceride lipase. The activated lipase then catalyses the hydrolysis of stored triglycerides into FFA and glycerol (Bjorntorp, 1991). The second mechanism by which caffeine may stimulate lipolysis is by a direct inhibition of PDE (Bellet et al., 1968; Wilcox, 1982) which contributes to an increased cellular concentration of c-AMP.

Both the time course for, and the magnitude of elevation of, increase in plasma FFA following caffeine ingestion are important considerations for the interpretation of ergogenic effects (Costill et al., 1978; Ivy et al., 1979). Whereas plasma caffeine concentrations peak about 1 hour following ingestion (regardless of dose), plasma FFA concentrations do not peak until at least 3 to 4 hours after ingestion depending on dose (Bellet et al., 1968, 1969; Essig et al., 1980; Ivy et al., 1979; Van Handel, 1979; Weir et al., 1987).

The problem with lipid utilisation during exercise is not the physical availability of fat as an energy source, but to bring the lipid to the site of oxidation in the muscle and have it used in the oxidative processes to furnish energy. If this can occur efficiently, then the limited carbohydrate (CHO) stores, which are essential for the needs of the brain, can be spared, and exercise prolonged. Training increases the capacity of skeletal muscle to

use fat as an energy source. An increase in fat metabolism during prolonged exercise has a glycogen sparing effect and as such improves endurance capacity. Fat cells increase their sensitivity to hormonal stimulation after training (Bjorntorp, 1991) and as a result mobilisation of fatty acid more closely matches utilisation.

Direct Measures of Muscle Lipid and Carbohydrate Use Indirect estimates of *in vivo* carbohydrate and lipid utilisation during exercise have implicated caffeine as a modulator of substrate mobilisation, uptake, and metabolism. Some information suggests that although caffeine may increase use of blood-borne FFA, it also affects muscle triglyceride use (Essig et al., 1980; McNaughton, 1986).

In one study, subjects cycled for 30 minutes at 70% of their maximum oxygen consumption (VO_2max). Caffeine (5 milligrams per kilogram of body weight; $mg.kg^{-1}$) was ingested 1 hour before exercise. Although there was an 18% increase in plasma FFA by the start of exercise, glycogen use was decreased by 42% and triglyceride was increased by 60% over control conditions. Estimates of substrate oxidation based upon respiratory exchange indicated that the subjects used 22% less carbohydrate during the 30 minutes of exercise following the caffeine ingestion. It was clear that caffeine altered use of endogenous muscle triglyceride (Essig et al., 1980).

These data support the suggestion that the 'sparing' of carbohydrate and improved endurance performance following caffeine administration are, at least in part, due to the altered rates of metabolism of intracellular substrate as opposed to increased uptake and oxidation of mobilised FFA. The latter may contribute to the modulation of muscle triglyceride pools.

CHAPTER TWO: CAFFEINE IN SPORT

Classification of Caffeine as a Doping Agent

Regardless of the ability of caffeine to induce a true ergogenic effect (Perkins and Williams, 1975), it has long been considered a doping agent that could enhance performance (Flinn et al., 1990). This led to its prohibition in athletic events. However, it was removed from the International Olympic Committee's (IOC) list of banned drugs in 1972. This was perhaps because research findings regarding its ergogenic effects were equivocal (Smith & Perry, 1992), that it is a constituent of many common beverages and, interestingly, that it is a constituent of a major Olympic sponsor's product. The IOC and the New Zealand Olympic and Commonwealth Games Association have since placed an upper limit on caffeine ingestion among athletes (Price & Weil, 1990; Wagner, 1990). Caffeine is restricted to 12 micrograms per millilitre ($\mu g.ml^{-1}$) of urine (New Zealand Sports Drug Association, 1994).

This level of caffeine is likely to be attained by injections or suppositories. Otherwise, to reach the upper allowable limit, Wagner (1990) suggests an athlete would have to ingest 6 cups in one sitting, an impractical exercise before competition. However, Birkett et al. (1990) showed that the limit of $12\mu g.ml^{-1}$ could be exceeded by some individuals with low to moderate caffeine intake. Therefore, consideration should be given to raising the limit, or alternatively, advising athletes to restrict intake to the equivalent of 2 to 3 cups of coffee per day.

The two most usual grounds for objecting to 'doping' are clearly set out in the Rules for Control of Banned Substances of the New Zealand Olympic and Commonwealth Games Association (NZOCGA, 1989, p. 17):

> There are some who consider that the use of these performance enhancing drugs (PEDs) is a development on par with improved dietary methods or improved technology and should not be penalised. The majority of sports administrators and athletes, however, appear to hold the view that the use of PEDs is a form of *cheating* ... Some of the PEDs are used by athletes in amounts far in excess of those used for therapeutic purposes and may be *dangerous to both physical and mental health* . (Italics added).

The objection could hardly be stated more clearly. The NZOCGA is opposed to the use of drugs, first, because it is not fair play, and secondly, because it may be damaging to health.

The three major categories of drugs used by athletes include: performance enhancement (ergogenic) drugs for the purpose of gaining athletic advantage; therapeutic drugs for specific medical indications; and pleasure drugs to alter mood and perceptions. These categories are not necessarily mutually exclusive. For example, a pleasure drug, such as caffeine, may both reduce anxiety in habitual caffeine users (therapeutic) and enhance performance by delaying muscle fatigue in athletes (ergogenic).

A number of anomalies have been identified in the IOC list of banned drugs (Shapiro, 1991). Firstly, caffeine is a drug that can enhance performance, yet it is only banned in high doses. Pseudoephedrine, an ingredient of many cold remedies, is a therapeutic rather than an ergogenic drug. That is, an athlete may be enabled to compete with a cold by taking the drug, however it is banned in any quantity.

Secondly, like the amphetamines, the ergogenicity of caffeine is a function of its dose-response curve (Ellinwood & Nikaido, 1987). In this regard, it is interesting that caffeine is the only stimulant treated in a dose-response manner in drug testing protocols. The imposition of a quantitative standard for caffeine as contrasted with an 'all-or-none' standard for, say amphetamine, reflects both the ubiquity and legality of caffeine in society (Graham & Spriet, 1991).

Thirdly, Duthel et al. (1991) query the validity of the upper authorised official limit for urinary caffeine in doping controls after confirming a finding of Delbeke & DeBackere (1984) that exercise decreases caffeine elimination. On these grounds they suggest that the nature of the sporting event as well as sex, weight, and sampling delay after exercise are all factors that argue against the utilisation of a unique standard.

Fourth, caffeine is the only one of 40 stimulants on the IOC banned substances list that athletes have unrestricted access to in New Zealand. The other 39 are listed as controlled drugs, prescription medicine, restricted medicine, or pharmacy-only-medicine (Hatherton, 1991). Of the other banned substances in the various doping classes (for example, Anabolic Steroids), all 55 registered medicines are restricted in New Zealand.

New Zealand introduced a policy against the use of PEDs in sport in 1989, and this is implemented by the Sports Drug Agency (NZSDA), established by statute in 1994. All sports reliant upon Hillary Commission funding must endorse the policy, and ensure its constitution covers testing and disciplinary action. Interestingly, the New Zealand Federation of Body Builders does not seek government funding and therefore is not obliged to test its athletes. Some consider this a blatant admission of guilt. The Task Force Report on the Misuse of Drugs in Sport (Hatherton, 1991) recommends that athletes in the other eleven sporting codes addressed in this paper be tested in the competition season for stimulants (including caffeine; Appendix 4).

Caffeine and its by-products can be measured in urine by spectrophotometric methods so that detection is relatively easy (Delbeke & Debackere, 1984). However, the exogenous or excessive administration of caffeine for doping purposes is difficult to prove, especially if the athlete is simultaneously using diuretics.

Ergogenicity of Caffeine - Methodological Issues

Two factors need to be considered when discussing the ergogenic effects of caffeine in sport. Firstly, 'ergogenic' must be defined. The dictionary defines an ergogenic agent as something that "tends to increase work output, especially by eliminating fatigue symptoms". Ergogenic aids usually refer to substances not found in normal diets that are taken to improve performance. Any drug that pharmacologically aids the muscular, circulatory, or respiratory system, is a potential ergogenic aid, and thus a potential doping agent. The IOC defines 'doping' as the:

> ... use by a competing athlete of any substance foreign to the body or of any physiological substance taken in abnormal quantity or taken by an abnormal route of entry into the body with the sole intention of increasing in an artificial and unfair manner his/her performance in competition. (cited in Hatherton, 1991; p18).

Secondly, there are methodological considerations that make interpretation of the data difficult. Determination of the ergogenicity of caffeine in a competitive setting is even more difficult than under *in vitro* or *in vivo* conditions. That no clear pattern of work enhancement exists is likely due to the following complicating factors:

- There is a dose-response effect (Ellinwood & Nikaido, 1987; Kirsch & Weixel, 1988; Waldeck, 1973);
- There are subject differences in sensitivity (Costill et al., 1987; Essig et al., 1980; Goldstein et al., 1965, 1969; Graham, 1978; Work, 1991) and tolerance to caffeine (Essig et al., 1980);
- There are differences in response between habitual users and nonusers (Bangsbo et al., 1992; Fisher et al., 1986; Goldstein et al., 1965, 1969; Kuznicki & Turner, 1986);
- There are species differences that make extrapolation of animal data tenuous (Second International Caffeine Workshop [SICW], 1980);
- Studies may have been poorly controlled (Schreiber et al., 1988b);
- The natural sympathetic response to exercise (Sutton, 1985) may mask similar responses caused by caffeine;
- There may be conflicting responses due to both direct and indirect actions (Sawyer et al, 1983);
- There may have been imprecision in the measurement of coffee and caffeine intake (Schreiber et al., 1988a&b);
- Studies may have employed too few subjects or questionable protocols, (Kirsch & Weixel, 1988);
- Some studies have reported the effects of caffeine *in situ* in muscle while others have reported its effects on the whole body (Bianchi, 1962);
- There may be differences in the type of task (eg. incremental/progressive load versus constant load/steady state) or the exercise intensity (Casal & Leon, 1985);
- There may be conflicting results due to subjects' fitness levels and diets (Murray et al., 1989; Schreiber et al., 1988a&b; Weir et al., 1987);
- The environmental conditions (Doubt & Hsieh, 1991; Sawyer et al., 1983) may not have been taken into account;
- There may have been differences in the athlete pool (eg. runners versus cyclists; Casal & Leon, 1985);
- Subjects' body composition and muscle mass relative to dose may not have been accounted for (Essig et al., 1980);
- Subject selection (male versus female; trained versus untrained; age) may have been poor;
- The exercise protocols used may have been different (eg. open-ended versus close-ended tests; Tarnopolsky et al., 1989); and
- The personality of subjects and time of day the drug is administered may not have been accounted for (Smith et al., 1991).

Three examples drawn from the above list of confounding factors illustrate how confusing it can be to draw firm conclusions regarding the ergogenicity of caffeine from the literature. Firstly, two studies involving human subjects (Costill et al., 1978; Ivy et al., 1979) gave male and female subjects of different body weights and caffeine intakes, absolute caffeine doses that, when expressed in relative terms to body weight, were some 50% greater for lighter, as compared to heavier, individuals. Moreover, the former study administered 5g of decaffeinated coffee in the control drink. Decaffeinated coffee has been reported to contain 1-5mg caffeine per cup (Table 1) as well as trigonelline and chlorogenic, tanic, caffeic, quinic, acetic, propionic, butyric, and valeric acids; ketones; acetonin; furfural; and other acidic carbonyl compounds. The pharmacological significance of these substances must be considered when discussing the effects of caffeine (Knowles, 1990).

A second example relates to the training effect. Foltz et al. (1943) reported that training overshadows any effect caffeine might exert. Moreover, Casal & Leon (1985) have suggested that caffeine has an effect only on non-elite athletes who have not had the benefit of increased lipolytic enzyme activity and mitochondrial density and size that endurance training causes.

Thirdly, controversy exists regarding the effects of caffeine because of failure in many studies to account for acute, chronic and occasional use, previous caffeine intake, length of abstention and other interacting factors. To ignore these factors will produce a distorted view of the effects of this drug on human sports performance and behaviour.

Caffeine and Sports Performance

Caffeine has long been consumed by athletes in the belief it will enhance performance (Butts & Crowell, 1985; Delbeke & Debackere, 1984; Grollman, 1930). However, caffeine may be interpreted to have positive, negative, or no effects, depending on the population segment (Kuznicki & Turner, 1985) and the type of exercise being investigated (Powers & Dodd, 1985; Slavin & Joensen, 1985). In addition, as discussed above, there are several design problems that make the literature concerning the ergogenic effects of caffeine suspect. Thus, this discussion of the effects of caffeine on sports performance, must be viewed with regard to those considerations.

Long Term Endurance Performance There is considerable evidence that caffeine enhances performance in submaximal long term exercise (Table 4). Athletes who ingest caffeine prior to an endurance performance can show increased lipolysis, mobilisation of FFA from adipocytes, and increased capacity for endurance exercise (Costill et al., 1978; Flinn et al., 1990; Ivy et al., 1979; McNaughton, 1986; Spriet et al., 1992). During endurance competition, the availability of muscle glycogen is a critical factor. The increased availability of fat mediated by caffeine ingestion exerts a glycogen-sparing effect by inhibiting enzymes (eg. phosphofructokinase) involved in carbohydrate metabolism (McNaughton, 1986; Spriet et al., 1992).

Early studies indicated that caffeine increases endurance times in fixed pace work bouts and decreases times in fixed-distance races. Ivy et al. (1979) found that during 2 hours of isokinetic (fixed pace) cycling, administration of 500mg caffeine significantly (7.4%) increased work production by trained cyclists compared to placebo. Oxygen consumption showed a parallel increase while perception of exertion remained unchanged. Estimates of substrate utilisation based upon oxygen consumption (VO_2) and respiratory exchange (RER) data have suggested that caffeine enhances the use of lipids during the last half of an exercise task. In a companion study (Costill et al., 1978), competitive cyclists were asked to ride at 80% of their maximal oxygen uptake (VO_2max) until voluntary exhaustion. Those who received caffeine continued for over 90 minutes, as opposed to 75 minutes for the control group.

Essig et al. (1980) reported a 42% reduction in glycogen use with a $5mg.kg^{-1}$ dose of caffeine during 30 minutes of cycling at 69% VO_2max. Plasma FFA were elevated 323% concomitant with a 40% decrease in muscle glycogen utilisation. The mechanism behind the increased rate of work production and total work output was suggested to be due to an enhanced lipid utilisation. The increased oxygen uptake may have been a result of caffeine directly stimulating metabolic rate as well as caffeine's positive effect on lipolysis and fat utilisation (Van Handel, 1983).

McNaughton (1986) similarly reported an increase in plasma FFA concentrations after ingestion of large doses of caffeine ($15mg.kg^{-1}$), enhancing aerobic running performance. These changes were associated with a 'sparing' of glycogen in red muscle and liver, and higher blood glucose levels following exhaustive exercise. There were no effects in fast-twitch white muscle, probably due to its low ability for fatty acid oxidation.

This glycogen sparing effect and an increased rate of lipolysis both contributed to an increased time to exhaustion (McNaughton, 1986). This effect is consistent with the fact that studies have not shown caffeine to be effective during short-term (less than 10 minutes) high-intensity exercise such as sprint running, which does not rely upon fat for fuel.

Giles & McLaren (1984) also reported significantly higher FFA levels, and lower respiratory exchange ratio (RER) and perceived exertion (RPE) values with caffeine ingestion ($5mg.kg^{-1}$) before and during a submaximal treadmill run. A decreased RER suggests a sparing of carbohydrate, or more specifically, increased FFA oxidation and lipid metabolism (Costill et al., 1978; Giles & MacLaren, 1984). Erickson et al. (1987) similarly found a 30% reduction in glycogen use following during 90 minutes of cycling at 65-70% VO_2max with a higher caffeine dose ($9mg.kg^{-1}$).

Recent studies (Graham & Spriet, 1991; Spriet et al., 1992) have also demonstrated ergogenic effects of caffeine in recreational cyclists and highly trained distance runners. Spriet et al. (1992) demonstrated that the glycogen-sparing effect occurs even at a relatively intense power output (80% VO_2max) but that this is confined to the initial 15 minutes of exercise. The delayed utilisation of glycogen early in exercise appears to enable athletes to cycle longer before glycogen depletion and exhaustion occur. It is now generally concluded that caffeine does improve endurance performance, possibly through increased mobilisation of FFA, which spares muscle glycogen for later use (Erickson et al., 1987). However, this carbohydrate-sparing effect of caffeine has been reported in cyclists even when their serum FFA levels were not greatly elevated by of caffeine (Costill et al., 1978; Essig et al., 1980; Ivy et al., 1979). Therefore, the actual mechanism by which caffeine improves endurance performance might be more complex, involving perception of effort and cellular mechanisms.

Concomitant with physiological changes during exercise are psychological, or perceptual, changes of exercise intensity. Research suggests that people exercising at physiologically equivalent work loads perceive their effort at varying intensities (Borg, 1970), causing them to either push themselves harder or to decrease their intensity of effort. Perceptual effort can be quantitatively rated and compared among individuals during exercise by the scale of perceived exertion (RPE; Borg, 1982).

Table 4
Summary of previous studies of caffeine and aerobic exercise performance*

Study	Exercise Mode, Intensity, Duration	Caffeine dose, Time Taken	Serum Free Fatty Acid Levels during exercise after Caffeine Ingestion	Effect of Caffeine on: Exercise Performance	Lipid Oxidation during exercise	Pre-test Nutrition on Exercise Patterns of Subjects
Bellet et al. (1968)	Rest	220mg	not studied	not studied	not studied	Rested for 1 day, 11h postprandial
Perkins & Williams (1975)	Progressive to exhaustion	4/7/10mg/kg 30min pre-exercise	not stated	no effect, RPE unchanged	no effect, RER unchanged	3-4h postprandial
Costill et al.. (1978)	Cycling, 80%VO_2max 75-90 min	330mg, 60min pre-exercise	.3-.5mmol/l, caffeine had no effect of FFA levels	+ve	+ve, -ve RER	Rested for 1 day, 6-12h postprandial
Ivy et al. (1979)	Cycling, 69%VO_2max 120 min	250mg,60min pre-exercise, 250mg during exercise	.3-1.0mmol/l, caffeine had no effect on FFA levels	+ve	+ve, -ve RER	Not stated
Essig et al. (1980)	Cycling, 65-75%VO_2 max 30min	5mg/kg, 60 min pre-exercise	.7-.5mmol/l, caffeine elevated pre-exercise & final FFA levels only	not studied	+ve, -ve RER & muscle glycogen utilisation	Normal mixed diet, 1 day rest, 12h postprandial
Powers et al. (1983)	Cycling, progressive to exhaustion	5mg/kg, 60min pre-exercise	.3-.2mmol/l, caffeine had no effect on FFA levels	no effect	no effect, RER unchanged	Reduced exercise for 24h, 6h postprandial
Giles & McLaren (1984)	Treadmill run, 75% VO_2max, 120min	5mg/kg, 60min pre-exercise	.8-1.8mmol/l, glucose ingestion decreased the effect	not studied, -ve RPE	+ve, -ve RER	Not stated
Casal & Leon (1984)	Treadmill run, 75% VO_2max, 45min	400mg, 60min pre-exercise	.7-.9mmol/l, caffeine increased FFA levels	not studied, -ve RPE	no effect, RER unchanged	No 'strenuous' run for 24h, normal diet, postprandial
Butts & Crowell (1985)	Cycling, 75% VO_2max, to exhaustion	250mg, 60min pre-exercise	not studied	no effect, RPE unchanged	no effect, RER unchanged	Limited for 24h, 8h postprandial
Gaesser & Rich (1985)	Cycling, progressive to exhaustion	5mg/kg, 60min pre-exercise	not studied	not studied	no effect, RER unchanged	Not stated, 12 h postprandial
McNaughton (1986)	Treadmill run, progressive to exhaustion	15mg/kg, 60min pre-exercise	.36-.81mmol/l, caffeine increased FFA levels	+ve, -veRPE	+ve, -veRER	Not stated
Weir et al. (1987)	Treadmill & road run, 75% VO_2max, 120min	6.5mg/kg, 3h pre-exercise	.4-1.0mmol/l, caffeine had no effect on FFA levels in CHO-loaded Ss	no effect, RPE unchanged	RER unchanged in CHO loaded subjects	Rested for 2 days, high CHO diet, 3h postprandial
Tarnopolsky et al. (1989)	Treadmill run, 70% VO_2max, 90min	6mg/kg, 60min pre-exercise	Caffeine increased FFA levels	no effect, RPE unchanged	no effect, RER unchanged	Rested for 1 day, 3h postprandial
Flnn et al. (1990)	Cycling, progressive to exhaustion	10mg/kg, 3-4h pre-exercise	.1 to .6 mmol/l, CA increased FFA levels	+ve, not studied	+ve, -ve RER	12h postprandial
Gastin et al., (1990)	Running, progressive to exhaustion	5mg/kg, 3.5h pre-exercise	not studied	no effect -ve RPE	not studied	Limited for 24h, 12h postprandial
Spriet et al. (1992)	Cycling, 80% VO_2max to exhaustion	9mg/kg, 60min pre-exercise	.24 to .46 mmol/l, CA increased FFA levels	+ve, not studied	+ve RER	Normal training, 2-4h postprandial
Wiles et al. (1992)	Treadmill run, 1500m high intensity	3mg/kg, 60min pre-exercise	not stated	+ve, -ve RPE	+ve, -ve RER	12h postprandial

*Adapted and extended from Weir et al. (1987)
The abbreviations used are: FFA = free fatty acid; VO_2max = maximal oxygen consumption; RER = respiratory exchange ratio; RPE = rating of perceived exertion; +ve = positive effect; -ve = decreased; Ss = subjects; nonsig = nonsignificant; h = hours; CA = caffeine

In the study of Costill et al., 1978, there were no differences in heart rate or VO_2, but the perceived exertion was lower with caffeine. This suggests that under some conditions perceived exertion scales may not serve as an accurate index of metabolic stress, and that caffeine may mask fatigue symptoms. As with amphetamines, caffeine is generally touted as improving alertness, concentration, reaction time and energy levels. People

taking the drug often feel stronger and more competitive (Wyndham et al., 1971). They believe they can perform longer before the onset of fatigue and that they can push harder in the face of greater physiological stress. This reduced perception of fatigue has potentially dangerous implications. A cyclist has already died from heat exhaustion while using amphetamine. However, this risk is unlikely to exist from caffeine use.

Rodrigues et al. (1990) also reported that caffeine ($5mg.kg^{-1}$) significantly lowers the subjective perception of effort (RPE) during a cycle ride to exhaustion but has no significant effect on endurance time, pulmonary ventilation, oxygen consumption, carbon dioxide extraction or respiratory exchange ratio. Powers & Dodd (1985) reported similar findings.

The findings concerned with the RPE may be attributed to metabolic and neuromuscular factors. Thus, the reduction in RPE scores with caffeine use could be due to a combination of retardation of muscle glycogen breakdown, altered neuronal stimulation enhancing recruitment of muscle fibres (Waldeck, 1973), or elevated intramuscular calcium concentration enhancing muscle contractility (Williams, 1991). It is probable therefore, that enhanced lipid metabolism and neuromuscular function combine to depress RPE scores throughout exercise when caffeine is ingested (Giles & McLaren, 1984).

Several groups have found no effect of caffeine on performance measured by the respiratory exchange ratio. Perkins & Williams (1975) found time to exhaustion on a cycle ergometer and RPE were no different to control time and RPE with three dosages of caffeine. This study directly contradicts the study of McNaughton (1986) even though the studies were quite similar. Gaesser and Rich (1985) found that ingestion of $5mg.kg^{-1}$ caffeine did not delay, and might even accelerate the onset of blood lactate accumulation. Others (Ben-Ezra & Vaccaro, 1982; Butts & Crowell, 1985; Falk et al., 1989; Gastin et al., 1990; Powers et al., 1983) found no significant differences in the time to exhaustion between caffeine trials and control trials. It should be noted that these studies used a progressive work task in favour of steady state exercise. Graded tasks are largely aerobic but they end with a maximal anaerobic effort to exhaustion.

Casal and Leon, 1985) reported that although plasma FFA were elevated significantly before exercise after caffeine ingestion, there was no indirect evidence of altered substrate utilisation or carbohydrate-sparing during

subsequent running. This showed that the psychological influence of consuming coffee, regardless of caffeine content, reduced perceived exertion, probably through a placebo effect (Fillmore & Vogel-Sprott, 1992).

Clearly, it remains to be established why caffeine ingestion has a marked and reproducible carbohydrate-sparing effect in cyclists independent of its effect on serum FFA levels whereas in runners, the carbohydrate-sparing effect of caffeine would appear to be dependent on its effect in elevating serum FFA levels (Giles & McLaren, 1984). However, even when caffeine ingestion elevates serum FFA levels in runners, this does not guarantee that there will also be carbohydrate sparing (Casal & Leon, 1985).

Reasons for the discrepancies between studies (evident in Table 4) are not readily apparent, although differences in type of exercise, the intensity, the caffeine dose, and subjects' diets may be responsible for such differences (Casal & Leon, 1985; Gaesser & Rich, 1985; Tarnopolsky et al., 1989).

The interaction of diet with the free fatty acid response to caffeine was first noted by Bellet et al. (1968). They showed that by itself, caffeine (250mg) caused blood FFA levels to rise to levels of .82 millimol per litre ($mmol.l^{-1}$) within 3 hours. However, when glucose was ingested simultaneously with caffeine, blood FFA levels were depressed for the first 2 hours after ingestion and rose to only .49 $mmol.l^{-1}$ after 3 hours. This finding was confirmed by Giles and McLaren (1984) who showed that glucose ingestion significantly diminished and delayed the FFA response to caffeine.

Weir et al. (1987) similarly reported that when athletes ate a high carbohydrate (CHO) diet and rested for 3 days ('carbohydrate-loading') before exercise, caffeine did not affect serum FFA levels. This raises two practical issues. First, it shows that the combination of carbohydrate-loading and a high CHO pre-race breakfast inhibits the effect of caffeine on lipid mobilisation. It follows that if the beneficial effect of caffeine in runners is dependent solely on caffeine's ability to stimulate lipid mobilisation, then ingesting caffeine immediately prior to competition is of little value, as such athletes with be carbohydrate-loaded and will have eaten a high CHO meal shortly before competition (Hellemans, 1991). Also, during an endurance event, athletes ingest a variety of glucose-electrolyte solutions to maintain fluid balance when sweat losses are high (Costill and Saltin, 1974), and to provide CHO for muscle and nerve energy demands (Erickson et al., 1987). These practices elevate serum insulin levels and would be expected to

interfere further with the effect of caffeine on lipid mobilisation. This argument would appear not to apply to cyclists in whom the carbohydrate-sparing effect of caffeine seems to occur independently of its effect on serum FFA levels. Alternatively, if the ergogenic effect of caffeine is actually via a stimulatory effect on the CNS, this argument does not hold.

The second practical point is the time course of the FFA response to caffeine. In most of the studies, exercise commenced 60 minutes after caffeine ingestion. Yet it is clear that the peak FFA response to caffeine occurs much later, usually 3 to 4 hours after caffeine ingestion (Bellet et al., 1968, Weir et al., 1987). Thus, if the ergogenic effect of caffeine depends on its lipid-mobilising effect, then it would seem logical that exercise should only commence when that effect is most pronounced. Alternatively, if the ergogenic effect is dependent on a stimulatory effect on the CNS, then exercise should commence when caffeine levels are greatest, which would appear to be within 3 hours of ingestion (Bellet et al., 1968).

These findings might explain why many of the previous studies (Costill et al., 1978; Ivy et al., 1979; Essig et al., 1980) have failed to show the expected degree of serum FFA elevation during exercise after caffeine ingestion. In at least two of these studies (Costill et al., 1978; Ivy et al., 1979), the athletes rested for the day before the study, and this may have led to a partial carbohydrate-loading effect. Alternatively, exercise may have commenced before the serum FFA levels had peaked (Weir et al., 1987).

With caffeine, athletes can achieve a higher percentage of their VO_2max without inducing metabolic acidosis and therefore perform a greater amount or intensity of work. That is, athletes have a higher lactate threshold which allows more work to be done at higher levels of lactic acid in the blood (Anselme et al., 1992; Costill et al., 1978; Essig et al., 1980; Flinn et al., 1990; Ivy et al., 1981; McNaughton, 1987). The alteration of the lactate threshold is consistent with the decreased RER since there is less acidity and less bicarbonate flushing (Spriet et al., 1992).

Besides substrate mobilisation, other effects induced by caffeine may prove beneficial in endurance activities. These include elevated VO_2 (Ivy et al, 1979), direct vasodilation, a reduced activation threshold (Waldeck, 1973), increased release of acetylcholine, increased release of Ca^{2+} ions from the sarcoplasmic reticulum, myocardial stimulation, facilitation of gastric emptying, diminution of somatic discomfort (Dickinson et al., 1984), and

hyperglycaemia (Cheraskin et al., 1967). However, hyperglycaemia should be prevented in some endurance sports such as ultramarathon events. Regular consumption of glucose during such events is advisable.

Some convincing evidence indicates that caffeine increases both the work output and endurance in long term exercise. The benefits are probably due to increased lipolysis and a decreased degradation of muscle glycogen. However, reports of studies on caffeine's effect on maximal aerobic power (VO_2max) and time to exhaustion in incremental or graded exercise appear to be equivocal. Future studies should attempt to standardise the exercise protocol, control for caffeine tolerance and use similar criteria to determine the attainment of VO_2max.

The general conclusion reached by most sports scientists and many athletes, is that caffeine, in spite of the conflicting evidence, has an effect during endurance type events but it is difficult to draw firm conclusions concerning caffeine's ergogenic potential during incremental work. The consensus is that even if caffeine is not ergogenic, taking it can do little harm.

High Intensity Prolonged Exercise Relatively little research has looked at the effects of caffeine upon high-intensity prolonged exercise, where the relative importance of the different physiological parameters required to produce a high level of performance and the physiological causes of fatigue can differ from those of endurance work (above) and short-term high-intensity exercise (below). Caffeine-induced increases in anaerobic exercise are likely due to neurologic stimulation, or a reduction in the sensation of fatigue. The problem within this area lies however, with the multitude of experimental paradigms employed by the researchers. In a recent, well-controlled experiment, McNaughton (1987) showed that large doses (10 and $15mg.kg^{-1}$) of caffeine given before incremental, exhaustive exercise improved performance significantly.

Using a motorised treadmill, Wiles et al. (1992) investigated the effects of the ingestion on 3 grams of caffeinated coffee on: the time taken to run 1500 metres; the selected speed at which athletes completed a 1 minute 'finishing burst' at the end of a high-intensity run; and respiratory factors, perceived exertion and blood lactate levels during a high intensity 1500 metre run. The dose used was one that would realistically be ingested by an athlete as part of their 'normal' diet without contravening doping

regulations. The results showed that caffeine: decreased the time taken to run the distance; increased the speed of the 'finishing burst'; and increased VO_2 during the high-intensity run. The study concluded that under the lab conditions, the ingestion of caffeinated coffee could enhance the performance of sustained high-intensity exercise.

High Intensity, Short Term Work Although much evidence suggests that caffeine may improve endurance performance, questions remain with regard its effects on brief, high-intensity exercise and neuromuscular function. Availability of fuels is not considered to be a factor for short-term intense work tasks. There are three levels at which short-term, high-intensity exercise performance might be influenced by caffeine: stimulation of the CNS; enhancement of neuromuscular transmission; and improvement of muscle fibre contractility.

There appear to be three types of short-term activity that might be influenced by caffeine. These include, activities in which speed and timing are important, activities which require strength and power, and activities which require short-term muscular endurance (Powers & Dodd, 1985).

Rapid movements Bradycardia and decreased reaction time can occur following administration of caffeine (Jacobson & Edgely, 1987), both of which could be of benefit in events requiring quick discriminative reactions and muscle control. Moreover, subjects feel more alert and active and have a decreased sensation of drowsiness (Goldstein et al., 1965, 1969; Smith et al., 1991). In addition, there can be a reduction in tremor or muscle tension as evidenced by altered EMG activity.

As previously discussed, caffeine stimulates the CNS and facilitates neuromuscular transmission. Both of these effects could result in reduction in the times required to initiate and complete rapid movements performed after receiving an external stimulus. The effects of caffeine on reaction and movement times (RT and MT, respectively) reported in the literature have been somewhat inconsistent.

However, despite the apparent inconsistencies, it is generally agreed that caffeine doses between 100 and 300mg facilitate RT and MT (Jacobson and Edgely, 1987; Lieberman et al., 1987; Silverman & Griffiths, 1992). Dosages outside this range tend to have varied or detrimental effects on RT (Jacobson and Edgely, 1987; Jacobson & Edwards 1990). Thus, moderate doses

of caffeine might improve performance in events that require quick reactions and rapid movements. Such events include running and swimming sprints.

Although caffeine may improve RT and MT, it also has negative effects on neuromuscular function in that it can impair hand steadiness and coordination (Jacobson et al., 1991, Kuznicki & Turner, 1985). This could lead to impaired performance in activities such as shooting and archery, where hand steadiness is important.

Maximal static and dynamic muscular contractions It is well documented that weight lifters and throwers use caffeine as an ergogenic aid (Lopes et al., 1983). Caffeine ingestion increases contractility of electrically stimulated muscle *in vitro* (Huidobro and Amenbar, 1945) and *in situ* (Lopes et al., 1983). However, when caffeine is used to modify performance in large-muscle short-term intense exercise requiring strength and power, the studies show essentially no effect (Asmussen & Boje, 1948; Bond et al., 1986; Butts & Crowell, 1985; Tarnopolsky et al., 1989; Williams, 1991).)

The study by Lopes et al. (1983) may explain, in part, these confounding results. They investigated the isometric tension developed during ulnar nerve stimulation with and without caffeine ingestion. At lower frequencies of stimulation, caffeine increased the tension developed while at high frequencies or at maximal voluntary contraction it had no effect. This suggests that under *in vivo* conditions, the sympathetic response to work stress is of such a magnitude that it masks the caffeine-induced alterations seen in *in vitro, in situ,* or resting *in vivo* studies (Van Handel, 1983). It is also possible that the homeostatic adjustments *in vivo* preclude effects on contractility seen *in vitro* or *in situ,* or that the dose relative to the muscle mass involved is too small.

During sustained, constant-load static exercise, times to exhaustion are generally not prolonged nor reduced by caffeine ingestion. Lopes et al. (1983) showed that maximal holding time of the adductor pollicus is not altered by caffeine. It should be pointed out that quantification of performance during sustained static contractions is difficult due to the large individual variability in times to exhaustion.

Dynamic muscular contractions are not only associated with maximal force that can be generated by the muscle but also the velocity at which the

muscle contracts. The product of these two properties is power output. Bond et al. (1986) found that caffeine ($5mg.kg^{-1}$) had no significant effect on power and peak torque generated by the knee flexors and extensors during isokinetic exercise. However, Anselme et al. (1992) showed that during short-term exercise, caffeine increases maximal anaerobic power. Caffeine did not change the highest work load (force). As anaerobic power is the product of force and velocity, caffeine ingestion must have increased the velocity. Anselme et al. (1992) also reported a higher exercise blood lactate concentration with caffeine at the highest exercise load compared to placebo. The mechanism for these effects appears to be related to Ca^{2+} release. This is in agreement with Falk et al. (1989) and Collomp et al. (1992).

Short-term muscular endurance Caffeine might also improve performance in sprint and strength types of activities where short-term muscular endurance is important (Anselme et al., 1992; Collomp et al., 1992). Unfortunately fewer studies have investigated this area, but caffeine might facilitate Ca^{2+} exchange at the sarcoplasmic reticulum and increase the activity of the sodium potassium pump, better maintaining the muscle membrane potential.

Early work concerning caffeine's effects on short-term muscular endurance and work capacity has yielded conflicting results (Foltz et al. 1942, 1943). More recent work indicates that caffeine has little or no effect on short-term muscular endurance and fatigue. During maximal exercise, both the rate and magnitude of fatigue are unaffected by caffeine (Collomp et al., 1992). These authors reported that specific training is necessary to benefit from caffeine-induced metabolic adaptations during exercise requiring a high anaerobic capacity. Caffeine (250mg) increased swim velocity over 100 metres in trained subjects but not in untrained subjects. This was possible because caffeine activity becomes more effective when intramuscular hydrogen ion (H^+) accumulation exceeds the intracellular buffering capacity (and anaerobic training enhances caffeine's buffering capacity).

Summary of Sport Performance Research

Athletes in a variety of sports reportedly use caffeine before athletic competition (Powers & Dodd, 1985). In addition, many governing bodies of sport consider caffeine a doping agent. However, the question of whether caffeine will improve athletic performance remains largely equivocal. Evidence concerning caffeine's effect on long term endurance performance

seems to suggest that caffeine may indeed enhance performance (Burke, 1980). The mechanism appears to be related to the increased availability of FFA for muscle substrate, causing a glycogen-sparing effect. The second type of activity where caffeine may have positive effects upon sports performance is in areas requiring increased alertness, quick reactions, rapid movements, and lowered anxiety levels. This has implications for athletes in events such as shooting or archery, where control, concentration, and perception are important. However, caffeine might also hinder performance in these activities that require hand steadiness.

Reports concerning caffeine's effect on VO_2max and performance during graded exercise are not in agreement. The reason(s) for these discrepancies are not clear. Evidence so far also suggests that use of caffeine as an ergogenic aid during exercise in which strength and short-term endurance are important has little effect. Acute caffeine ingestion does not seem to increase maximal voluntary contractions nor maximal power output nor delay fatigue during anaerobic exercise. Thus, use of caffeine to improve performance in activities requiring strength and short-term endurance (such as weightlifting) seems unwarranted.

There is still much to be learned about the physiological effects of caffeine in humans. Future research should take into account the methodological concerns listed previously.

Health Consequences of Caffeine

Concerns regarding caffeine's potential adverse health effects, both physical and psychological, have long been voiced (Benjamin et al., 1991; Wadler & Hainline, 1989) and scientific research abounds on the subject. health. Given its wide consumption, the health hazards are few, but caffeine has been attacked for a statistical association with various diseases (Ashton, 1987; Knowles, 1990). However, fears of a causative role in urinary tract and pancreatic cancer (Knowles, 1990), spontaneous abortion, teratogenesis, fibrocystic breast disease (Phelps & Phelps, 1988; Davis, 1990), have largely been dispelled (Ashton, 1987). Epidemiological evidence indicates that caffeine does not increase the incidence of hypertension or myocardial infarction (Grossarth-Maticek & Eysenck, 1991; Watson, 1988). Excellent reviews on the health consequences of caffeine can be found in Curatolo & Robertson (1983), Davis (1990), and Stavric (1988).

PART TWO

THE PRESENT STUDY

CHAPTER THREE: RATIONALE FOR THE PRESENT STUDY

The previous literature review highlights the extent to which studies regarding the ergogenic effects of caffeine provide equivocal results. The overuse, misuse and abuse of drugs is a major problem in society (Weinhold, 1991) and in sport (Waddington & Murphy, 1993), so it is not surprising that athletes, of any calibre, are involved. Professional athletes found to be using illegal drugs receive a great deal of media attention, but little is reported about the much greater number of amateur athletes who are involved, unless they are disqualified because of positive drug tests. It is impossible to arrive at a precise estimate of the extent of illicit drug use in contemporary sport for those who use performance-enhancing drugs inevitably seek to do so without being detected. This is because many drugs are prohibited by the IOC and other sporting bodies, and because the possession of such drugs may also constitute a criminal offence. However, an indication of the extent of drug abuse in sport can be derived from urine samples analysed by IOC-accredited laboratories. In 1989, 1206 (2.3%) of 52371 tests returned positive results (Hatherton, 1989). Anabolic steroids and diuretics appear to be the most commonly abused drugs in sport.

Attempts to control, if not eliminate, the illegal use of drugs among athletes has gained momentum in recent years. Sports organisations and government bodies have attempted to examine the extent of drug use, while providing increasingly harsh penalties. However, preoccupation with illicit drug use by athletes has diverted the attention from the abuse of licit drugs like caffeine. Quantitative research into the extent of caffeine use is by athletes is lagging, perhaps due to its ubiquitous nature in our society. Most of the accumulated evidence in this area is anecdotal (Anshel, 1991). Pigeonholing drugs into one category or another has tended to obscure the reasons for their abuse, as well as the consequences of that abuse. Even categorising drugs as recreational, therapeutic, and performance-enhancing suggests that the distinction between categories is well defined, yet pharmacological principles indicate otherwise. For example categorising caffeine as a recreational drug does not account for its effect of performance.

Furthermore, this literature review has highlighted that correlations between laboratory experiments and competition performance may not be close enough to justify banning caffeine solely from results of such tests. Consider caffeine's effects on the performance of a cyclist. In the lab setting, an athlete who uses caffeine can perform better doing an ergometer cycle to

exhaustion. How does that translate to the competitive performance of the same cyclist. Is she or he faster? Are his or her tactics and strategies better? On the other hand, if perception is a person's reality, does mere belief that a drug is ergogenic make it a performance-enhancing drug? Certainly it is an issue to be considered when discussing the ergogenicity of caffeine.

Education is necessary to communicate to all sports medicine professionals (as well as athletes and their families and coaches), the realities about the benefits and inherent dangers of caffeine. Such information may prevent athletes from harming themselves, and assist their sports performance. However, to ascertain whether education of athletes is warranted (for example, in financial terms) or necessary, it may make more sense to address the issue of the extent of caffeine abuse by New Zealand athletes. This project was therefore developed to examine the seriousness and prevalence of caffeine use by New Zealand athletes.

Specifically, this study examines the extent of caffeine use by athletes in this country and extends a number of research findings from college students in the United States to a pool of New Zealand athletes representing 12 sports. Bradley and Petree (1990) used the expectancies' paradigm developed in alcohol research to study caffeine consumption and signs of caffeinism in college students.

Interest in caffeine is motivated by clinical pragmatism. Caffeine consumption is ubiquitous in our society, and the potential adverse effects of caffeine consumption by athletes have recently become the focus of attention. Not only is caffeine intoxication (or caffeinism) a recognised 'mental disorder', but caffeine is also banned at high levels in the sports arena. Moreover, caffeine abuse has been cited as a 'problem' amongst athletes but there has been no survey research indicating how much of a problem it really is. Caffeine-induced symptoms are of clinical significance in and of themselves. Caffeinism can also complicate differential diagnosis of other psychiatric disorders, such as anorexia nervosa, a common complaint among many female athletes.

Expectancies regarding caffeine-enhanced performance

In the present study, the role of expectancies regarding the performance-enhancing effects of caffeine as a motivational factor was investigated. Since the expectancies paradigm from alcohol research was found to be valuable

in predicting patterns of problem caffeine consumption among college students (Bradley & Petree, 1990), it is potentially useful to extend the caffeine expectancy measure to athletes in order to predict the extent of caffeine consumption and the occurrence of caffeinism symptoms.

For many people, caffeine's effects reflect its stimulant properties. These effects include enhanced psychomotor performance, alertness, and relief of fatigue (Jacobson & Edgely, 1987; Rodrigues et al., 1990). There is some experimental evidence that college students expect that caffeine will enhance their performance. Fillmore and Vogel-Sprott (1992) found that placebo effects of caffeine determined college students' mood and performance on motor performance, such that those who expected enhanced mood or performance experienced the same while those who expected impaired mood and performance showed impairments. Page (1987) reported that college students who prefer to drink caffeine-containing drinks maintain different perceptions about the positive and negative consequences of drinking caffeinated drinks from those who do not prefer caffeinated drinks. Kirsch and Weixel (1988) observed an inverted-U shaped 'dose'/response curve in the performance of college students who were deceived to believe that they were consuming varying levels of caffeine and the occurrence of caffeinism symptoms. The results clearly point to a behaviourally potent expectancy of caffeine's enhancement of performance.

Gilliland and Andress (1981) studied the relationship between regular coffee consumption and the incidence of caffeinism in a nonclinical population (159 college students). The survey battery included the State-Trait Anxiety Inventory and the Beck Depression Scale. The moderate (one to five cups per day) to high (five or more cups per day) coffee consumers had significantly higher trait anxiety and depression scores than the abstainers and the low (less than one cup per day) users. These anxiety levels were subclinical, but the authors suggest that they may indicate significant and stressful differences in such factors as sleep patterns and nervousness. These individuals may be more likely to experience clinical levels of anxiety in stressful situations (such as the competitive sports arena) than low users. The high consumers also reported significantly higher levels of caffeinism symptoms, higher frequencies of psychophysiological disorders, and lower academic performances.

The aim of the present study was to extend the scales of Bradley and Petree (1990) and of Page (1987) reflecting college students' beliefs about

caffeine's enhancement of performance to an athlete pool. Based on prior findings linking positive caffeine expectancies, high caffeine consumption, and negative caffeine outcomes (caffeinism), it was hypothesised that the extent of endorsement of personal expectancies of performance-enhancing properties of caffeine-containing beverages would be positively related to level of caffeine consumption and number of signs of caffeinism reported by each subject. A further scale was developed to examine the extent of endorsement of expectancies specific to athletic performance.

In additional analyses, as an analogue to DSM-IIIR classification, subjects were divided into those reporting five or more signs of caffeine intoxication (caffeinism 'syndrome present') and those reporting fewer than five signs (caffeinism 'syndrome absent'). These groups were then compared, hypothesising that the syndrome-present group would have significantly higher caffeine intake and more performance-enhancement expectancies regarding caffeine consumption than the syndrome-absent group.

A third hypothesis was tested with relation to athletic identity or the degree to which an individual identifies with the athlete role. Brewer et al. (1993) developed a reliable and valid measure of athletic identity, the Athletic Identity Measurement Scale (AIMS). These authors identify a number of potential benefits of a strong athletic identity. However there are a number of potential costs of strong athletic identity. It is possible that a strong athletic identity may prompt individuals to engage in a sport or exercise activity to the extent of jeopardising their physical health. According to the authors: "excessive training, participating in sport while injured, and other such behaviours may in some cases negate the potential health/fitness benefits of a strong athletic identity". Therefore, another aim of this study was to examine the relationship between athletic identity and the use of caffeine as an ergogenic agent. The hypothesis tested was that athletes who identify strongly with the athlete role are more likely to engage in drug-taking behaviour than athletes who have a weak athletic identity. Furthermore, athletes who compete at a national or international level are more likely to have a strong athletic identity than those participating at local community or regional level. It is intended that these assumptions would identify any differences in caffeine consumption between top class athletes and recreational athletes.

CHAPTER FOUR: METHODS

This study tested the assumption that athletes with high levels of caffeine consumption have greater expectations of caffeine-enhanced performance and more caffeinism symptoms than athletes with low levels of caffeine consumption. Chapters 1 and 2 addressed some of the physiological and psychological effects of caffeine, and how these might affect athletic performance. The design of this study was chosen to replicate and extend some research conducted on college students to determine whether or not this research might be applied to an athlete sample. This study and the research protocol were approved by the University of Canterbury Human Ethics Committee.

Subject Contact

A letter (Appendix 5) was distributed to 12 National Sports Organisations (NSOs) whose addresses appeared on the NSOs Contact Address List (Hillary Commission, 1994). This letter was addressed to each respective president and requested their assistance with the delivery of questionnaires to athletes within their organisation. Only 3 of the NSOs were able to offer their assistance (Appendix 6). Canterbury regional sporting bodies, which appeared in the Canterbury All-Sports Sports Diary (Summer 1994-1995), were contacted and asked to distribute questionnaires to athletes of the nine, as yet unaccounted, sports.

Because of the Privacy Act (1994), athletes were not able to be contacted personally. Instead, each distributor received a pack of 30 questionnaires to be sent to a representative sample of athletes affiliated to their organisation in 1994. Each envelope had been pre-stamped and sealed. All that was required of each distributor was to address and mail each of the 30 envelopes to a 'representative sample' of athletes. It was requested that they be sent to athletes over the age of 18, both male and female, who represented the various levels of participation in that sport.

One week after delivery of the questionnaires to the distributors, they were contacted by the researcher to ensure they had made delivery of all their allocated envelopes. All 360 questionnaire packs had been mailed out or delivered by hand to individual athletes.

A copy of the questionnaire was provided to each NSO or regional representative for their respective organisation's records. Each organisation was also provided with summary findings once the results had been collated, and interested parties were invited to discuss with the researcher anything relating to the study.

Choice of sports

There are several sports in which caffeine is not permitted because it is considered to artificially assist an athlete's performance. Each sport in this study, with the exception of body building, was listed in the Task Force Report on the Misuse of Drugs in Sport (Hatherton, 1991) and had a recommendation attached to that listing that athletes in these sports be tested for stimulants during competition. The 12 sports were chosen because they reflected the range of activities in which an athlete's performance might be affected by caffeine in different ways:

Athletics	Endurance capacity
Archery	Anxiety, reaction time, fine motor coordination
Body Building	Pre-competition fat-loss, 'making weight'
Boxing	'Making weight', strength and power
Cycling	Endurance capacity
Fencing	Endurance capacity, anxiety, reaction time
Ice Racing	Strength and power, endurance capacity
Olympic Wrestling	'Making weight', strength and power
Power Lifting	'Making weight', strength and power
Rowing	Endurance capacity, 'making weight'
Target shooting	Anxiety, reaction time, fine motor coordination
Triathlon	Endurance capacity

Caffeine is used by athletes in endurance sports (Powers & Dodd, 1985) such as running, cycling, triathlon and rowing. It is also used by athletes who must 'make' weight categories, abused for its reported diuretic and thermogenic effects. Examples are wrestling, boxing, body building and rowing. Participants in these sports often reduce weight at the time of weigh-in by dehydration (Clement, 1991). Caffeine ingestion is a means to this end, as well as providing purported endurance benefits where there is an unwillingness to take calories for fear of not making weight. Body builders also require a lean physique for aesthetic purposes, (leanness accentuates muscularity). Caffeine may be useful in archery and target shooting where decreased reaction time and reduced anxiety are desired. It may also be useful in sports requiring strength and power and has been used by weightlifters as an ergogenic aid for this purpose (Bond et al., 1986).

Procedure

Subjects for this study were all athletes affiliated to one of 12 regional or national sports organisations (Appendix 7). They were informed that they would incur no postal expenses, and that individual scores and identities would remain confidential. The subjects were also told that their respective NSOs would receive a summary of the results at the end of the study, but that details of their individual results would not be disclosed. Subjects interested in receiving such a summary were invited to request information directly from the researcher. Athletes were encouraged to contact the researcher for any reason relating to the study.

Of 360 questionnaires sent out, 185 were returned correctly completed. One questionnaire was returned, opened but not filled out. Four additional questionnaires were returned unopened, stamped "Return to Sender" by New Zealand Post because they were either insufficiently addressed (by the appointed distributor of questionnaires), or because the subject was no longer at the address. Two questionnaires were received after the final report had gone to print. These subjects noted they had been overseas, training and competing, and were very enthusiastic about participating in the study.

Each questionnaire pack contained an introductory letter (Appendix 8) and a consent form (Appendix 9). A pre-addressed post-paid envelope was included to facilitate return of completed forms to the researcher. Subjects were also forwarded the following items:

Demographic questionnaire	(DEMO-Q; Appendix 10)
Caffeinism symptoms questionnaire	(CAFF-SX; Appendix 11)
Perceived consequences of caffeine	(EP-CAFF.1; Appendix 12)
Perceived consequences of caffeine in sport	(EP-CAFF.2; Appendix 13)
Sport specific questionnaire	(SPORT-Q; Appendix 14)
Athletic Identity Measurement Scale	(AIMS; Appendix 15)
Caffeine Intake Questionnaire	(T-CAFF; Appendix 16)

Subject Gifts Subjects were gifted a one-month membership card to Les Mills World of Fitness in Christchurch (valued at $45; Appendix 17) regardless of whether or not they responded. All subsequent respondents were invited to be included in a draw for a 6-month membership at the same fitness centre (valued at $295) and were provided with an entry form for this purpose (Appendix 18). It was hoped that this would provide an incentive for participation.

Follow-up No correspondence was entered into with non-respondents (those who had not returned their questionnaires within 2 months). The feasibility of a second mailing of questionnaires with another stamped return-addressed envelope to solicit co-operation, was outside the budget constraints of this research. For this reason, a return of just over 50% (n=185) had to considered acceptable.

Consent Athletes were requested to sign and date a consent form to comply with Human Ethics regulations. Consent was obtained from subjects after informing them in writing that the study involved completing a questionnaire that was constructed to assess their caffeine consumption and their perception of caffeine's effects upon them.

Method of Data Collection

Questionnaires The scales were designed to obtain self-report data on a number of variables. Ease and rapidity of questionnaire completion were considered in the selection of scales. The scales described below were formatted as a questionnaire for subjects to complete. Each questionnaire was coded with an individual identifier number, 3 digits long, which appeared on every page except the consent form. The questionnaires were numbered 001 to 360.

The first page of the questionnaire was an introductory letter to subjects outlining the purpose of the research and requesting their assistance. Once the questionnaires had been returned, all consent forms and prize-draw entry forms were separated from the questionnaires to preserve each subject's anonymity. The entry forms were placed in a sealed box, and a winner was drawn on location at Les Mills World of Fitness. The winner was notified by mail by a Les Mills employee.

Scales

Demographic Questionnaire (DEMO-Q) This questionnaire was designed to elicit information about each subject's gender, age, income, marital status, ethnic identity and education (Appendix 10).

Caffeinism Symptoms Questionnaire (CAFFSX) This is a 10-item self-report measure of subjects' tendencies to experience various signs after consuming a caffeinated beverage (Appendix 11). Subjects were asked to rate

the extent to which they experienced each symptom on a three-point scale (1=never; 2=sometimes; 3=often) when they consumed caffeinated beverages. The questionnaire items were based on DSMIII-R 'caffeinism/ caffeine intoxication' signs. Previous research showed that a variation of this questionnaire (Bradley & Petree, 1990) was sensitive to caffeine. Sample items are: "tremor" and "heart palpitations".

For later analyses, these items were re-coded so that they received no point for each symptom they 'never' experienced, and one point for an endorsement of a symptom (that is, those they 'sometimes' or 'often' experienced). Thus, scores ('CAFFSX11') can range from 0 to 10, with higher scores indicating a higher endorsement of caffeinism signs. Then, as an analogue to DSMIII-R classification ('INTOX'), subjects whose scores were 5 or greater were identified by the 'caffeinism syndrome present' label. Those scoring below 5 were classified as 'caffeinism syndrome absent'. This measure was included to assess the extent to which subjects' endorsement of caffeinism signs and caffeine intake influenced their responses to the 'caffeine expectancies' scales (below). Subjects were also asked to rate the extent to which they purposely chose to drink non-caffeinated beverages on a 4-point scale (1=always, 2=usually, 3=sometimes, 4=rarely), and whether the last drink they consumed was caffeinated or caffeine-free.

Caffeine expectancies (EP-CAFF.1) The rating instrument selected to measure athletes' expectations of caffeine was based on a scale used by Page (1987), which consists of 26 statements concerning the possible effects of caffeine. The statements reflect 10 beliefs about the perceived positive effects of drinking caffeinated beverages and 16 beliefs about the perceived negative effects of drinking caffeinated beverages (Appendix 12). Sample items are "Caffeine helps people stay alert" and "Caffeine makes people nervous and anxious". Subjects were asked to indicate their beliefs about the characteristics of caffeine by answering 'Yes' (coded as 1), 'No' (coded as 2), or 'Unsure' (coded as 3). In further analyses the 'unsure' responses were ignored, the 'yes' responses re-coded as 1, and the 'no' responses re-coded as 0.

Caffeine expectancies in sport (EP-CAFF.2) This measure assesses subjects' beliefs about the characteristics of caffeine as they relate to sport. A scale was developed using 13 belief statements about the perceived effects of using caffeine in sports (Appendix 13). The item from the pool was elicited from various research findings regarding the potential worth of caffeine as a

performance-enhancing drug. Sample items are: "Caffeine can be of benefit in sports requiring endurance" and "Very high doses of caffeine can impair sports performance". Subjects were asked to indicate whether they maintained each belief by answering 'Yes' (coded as 1), 'No' (coded as 2), or 'Unsure' (coded as 3). In further analyses the 'unsure' responses were ignored, the 'yes' responses re-coded as 1, and the 'no' responses re-coded as 0.

Sport Specific Questionnaire (SPORT-Q) A sport-specific questionnaire (Appendix 14) was designed to elicit information about each subject's sport, their main reason for participating, and the number of years they had been competitive. Subjects also answered an item requesting information regarding their highest level of athletic involvement (classifications were local community, regional, national, and international). This questionnaire also asked if they had ever used caffeine in sport for its performance-enhancing effects, even if it meant they risked disqualification as a result of a positive urine test being returned after drug-testing.

Athletic Identity Measurement Scale (AIMS) The rating instrument selected to measure the extent to which subjects identified with the athlete role was the AIMS (Appendix 15) because it is considered a reliable and valid measure of athletic identity (Brewer et al., 1993). It consists of a list of 10 five-point Likert items anchored by 1 (strongly agree) and 5 (strongly disagree). Sample items in the AIMS scale are: "I consider myself an athlete" and "I would be very depressed if I was injured and could not participate in sport". The item pool was designed to be representation of the social, cognitive, and affective aspects of athletic identity. Scores can range from 10 to 50, with lower scores reflecting a stronger identification with the athlete role. For the purpose of this study, subjects whose scores fell below 25 were classified as high in Athletic Identity, whereas those equal to or above the 25 were operationally defined as low in Athletic Identity.

Daily Caffeine Intake Questionnaire (TCAFF) Also included among the instruments was a caffeine intake questionnaire (Appendix 16). Average daily total caffeine consumption (in milligrams) was calculated as the sum of caffeine per day using estimates by various authors on the caffeine content of commonly consumed beverages (Table 1). These estimates are known to vary widely, however, according to the brand and brewing method of each beverage. Note that the 'Average' value reported in said table was used to ascertain caffeine consumption.

Although consumption of the most common caffeine-containing substances was monitored (for example, coffee, tea, colas, chocolate, NoDoz, and other drugs), coffee and tea overwhelmingly accounted for the majority of caffeine ingested by any one subject, with colas and chocolate making up the balance. Interestingly, no drugs contributed to the caffeine intake of any of the subjects. The level of caffeine consumption ('TCAFF') was coded according to the following criteria:

0 - 99mg daily caffeine intake was coded as 0
100 - 199mg daily caffeine intake was coded as 1
200 - 299mg daily caffeine intake was coded as 2
300 - 399mg daily caffeine intake was coded as 3
400 - 499mg daily caffeine intake was coded as 4
500 - 599mg daily caffeine intake was coded as 5
600 - 699mg daily caffeine intake was coded as 6
700 - 799mg daily caffeine intake was coded as 7
800 - 899mg daily caffeine intake was coded as 8
900 - 999mg daily caffeine intake was coded as 9
above 1000mg daily caffeine intake was coded as 10

In further analyses level of caffeine consumption was further defined. Subjects were assigned to three groups ('INTAKE') based on their average daily caffeine consumption. Low caffeine intake was operationally defined as less than or equal to 200mg per day; moderate caffeine intake was defined as between 200 and 400mg per day; and high caffeine intake was defined as greater than 400mg per day.

Dependent Variables The dependent variables were each calculated as follows: A score was obtained based on the number of positive EP-CAFF.1 scale items endorsed by each subject. In order to replicate the study of Bradley and Petree (1990) the dependent variable of the first hypothesis is confined to analysis of the following 6 scale items: 13, 15, 19, 23, 25, 26. The contents of this scale ('EXPECT' motives) defines caffeine use in terms of the expected 'functions' which this drug is intended by the user to serve. These functions reflect caffeine's central nervous system stimulating effects.

In order to replicate the study of Page (1987), a score ('POSITIVE') was obtained based on the total number of positive EP-CAFF.1 scale items endorsed by subjects. This included the following items: 1, 3, 4, 5, 13, 15, 19, 23, 25, 26. Another score ('NEGATIVE') was obtained based on the total

number of negative EP-CAFF.1 scale items endorsed: 2, 6, 7, 8, 9, 10, 11, 12, 14, 16, 17, 18, 20, 21, 22, 24. These item numbers are listed in Appendix 20.

In order to examine sporting enhancement beliefs, a score ('ENHANCE') was obtained based on the total number of positive EP-CAFF.2 scale items: 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11 and 13. The individual items are listed in Appendix 21. The twelfth item was not included in the 'ENHANCE' score because it was coded in the opposite direction to the other statements.

Subjects were currently competing in one of twelve sports (in order of descending frequency): fencing (11.4%; n=21); cycling (10.8%; n=20); rowing (10.3%; n=19); triathlon(9.7%; n=18); body building (9.7%; n=18); wrestling (8.6%; n=16); athletics (8.6%; n=16); power-lifting (8.1%; n=15); archery (8.1%; n=15); ice-racing (7.0%; n=13); shooting (6.5%; n=12); and boxing (1.1%; n=2). Data from the male and female subjects and across sports were combined for most analyses after ANOVA revealed no significant differences for gender on either caffeine intake or sport (see Results Section).

Data Analysis

The data were analysed using the Statistical Package for the Social Sciences (SPSS for Windows) by descriptive statistics, analysis of variance (ANOVA), multivariate analysis of variance (MANOVA), multiple regression and chi-square analysis, depending on the variable distribution and research question. For all statistical tests, effects were considered significant for $p \leq .05$.

CHAPTER FIVE: RESULTS

Introduction The aim of this study was to determine caffeine consumption, expectations of caffeine-enhanced performance and caffeinism symptoms in a sample of New Zealand athletes. It was also considered important to measure the extent to which subjects who identify strongly with the athlete role engage in drug-taking behaviour, specifically caffeine 'abuse'. This chapter describes the characteristics of the sample, and presents the results of the data analyses.

Demographic Characteristics A total of 185 subjects completed the questionnaire and all of these were considered valid. Subjects were excluded from analyses when data were missing on the variables concerned. High caffeine consumers (>400mg per day) accounted for 8.6% (n=16) of returns; 23.8% (n=44) were moderate caffeine consumers (200 to 400mg); and 67.6% (n=125) were low caffeine consumers (<200mg per day). Athletes who identified strongly with the athlete role made up for 51.4% of the sample.

Age Almost half of the subjects (45.9%) were aged between 18 and 25 years. 17.3% represented the 26 to 30 age-group, 12.4% were aged 31 to 35, 7.6% were aged 36 to 40, 9.2% were aged 41 to 45, 4.9% were aged 46 to 50, and 1.6% were over the age of 50. Two subjects did not divulge their ages. Sixty males (32.4%) and 124 females (67%) responded to the questionnaire.

Education In terms of the highest education level achieved, 15.1% had gained school certificate, 25.4% had achieved sixth form certificate or university entrance, 14% had completed Form 7, 19.5% had a university qualification, 13.5% had a trade certificate, and 12.4% declined to answer.

Income The annual personal incomes reported by subjects are as follows: 31.4% earned less than $10000, 20.5% earned between $20000 and $30000, 21.1% earned between $30000 and $40000, 12.4% earned between $40000 and $50000, 5.4% earned between $40000 and $50000, and 7.6% earned over $50000. Those who did not provide personal financial information constituted 1.6% of the sample. The following are the statistics reported for annual household incomes: 7% earned less than $10000, 5.4% earned between $20000 and $30000, 15.7% earned between $30000 and $40000, 17.8% earned between $40000 and $50000, 15.1% earned between $40000 and $50000, and 31.9% earned over $50000. Seven percent did not provide household financial information.

Relationship and Family Status The majority (58.7%) described themselves as single, 28.1% were married, 7.6% were in de facto relationships, 5.4% were divorced or separated. One subject (.5%) did not disclose relationship information. Most subjects (73.0%) had no children, 3.2% had one child, 10.3% had two children, 9.2% had 3 children, and 3.2% had four or more children. Two subjects declined to comment.

Ethnic Origin When asked to describe their ethnic origin 93% classified themselves as New Zealand European, 1.1% as New Zealand Maori, 1.1% as Polynesian, and 0.5% as Asian. A total of 3.8% classified themselves as identifying with an 'other' ethnic origin, and one respondent did not complete this section of the questionnaire.

Supplementary Correlations and ANOVAs No significant correlations were found between the demographic variables (income, education, relationship status, number of children, and ethnic origin) and caffeinism symptoms or caffeine intake. However, analysis of variance (ANOVA) revealed a significant difference between caffeine intake ('TCAFF') groups on the basis of age [$F(5,178)=4.021$, $p<.01$]. Analysis of variance revealed a nonsignificant difference between caffeine intake groups on the basis of gender [$F(1,182)=.04$, $p=.84$ (n.s)], and a significant F-ratio was revealed for the effect of gender on 'CAFFSX11' (number of caffeinism symptoms endorsed) [$F(1,184)=5.70$, $p<.05$], with females endorsing more signs than males. No significant differences were found for the effect of gender intake on sport [$F(1, 183)=.90$, $p=.34$ (n.s)], nor of sport on caffeine intake ('TCAFF') [$F(11,183)=1.52$, $p=.13$ (n.s)].

Internal Consistency The internal consistency, using Cronbach's coefficient alpha, of the respective expectancies scales are as follows: 'EXPECT' (.6872), 'POSITIVE' (.6950), 'NEGATIVE' (.9332), 'ENHANCE' (.8495), 'AIMS' (.8689) and 'CAFFSX11' (.7880). These reliability coefficients were judged it to be adequate for the present study, that is, all alphas reached at least the .05 level of significance.

Relationship between Caffeine Consumption and Caffeinism Symptoms Figures 5 shows the distribution of subjects by their average daily caffeine intake. When subjects were further classified into three caffeine intake groups, 35 subjects reported being 'high' daily caffeine consumers, 79 were 'moderate' consumers, and 70 comprised the 'low' consumption group.

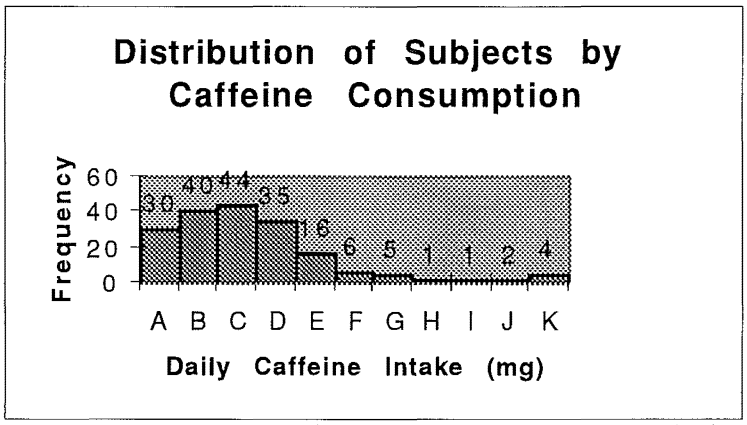


Figure 5 - Distribution of subjects by their average daily total caffeine consumption. Key: A 0-99, B 100-199, C 200-299, D 300-399, E 400-499, F 500-599, G 600-699, H 700-799 I 800-899, J 900-999, K Above 1000 milligrams.

Figure 6 displays the distribution of subjects by the number of caffeinism signs they endorse. Subjects classified as 'caffeinism syndrome present' (that is, those endorsing 5 or more of the DSMIII-R symptoms) number 46, and those labelled as 'caffeinism syndrome absent' number 139.

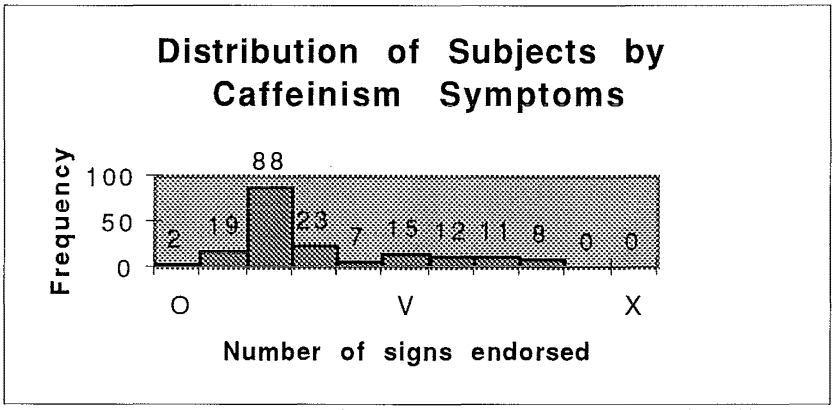


Figure 6 - Distribution of subjects by the number of caffeinism symptoms endorsed by each. Those endorsing 5 or less are classified as caffeinism syndrome absent, those endorsing more than five are classified as caffeinism syndrome present.

Caffeinism data The association between the amount of caffeine consumed ('INTAKE') and the caffeinism status of subjects ('INTOX') is presented in Table 5. The caffeinism syndrome present group was composed of 63% low caffeine consumers, 26.1% moderate caffeine consumers, and 10.9% high caffeine consumers. The constitution of the syndrome absent group is as follows: 68.4% are low caffeine consumers, 23.5% are moderate caffeine consumers, and 74.7% are high caffeine consumers. The three levels of caffeine consumption ('INTAKE') were subdivided by the caffeinism status of subjects ('INTOX'). 3 X 2 chi-square ($\chi 2$) analysis [$\chi 2(2,181)=.54$, $p<.50$ (n.s)] yielded a non-significant contingency

coefficient (p=.76). This indicates that subjects' caffeine intakes and their caffeinism status (present or absent) are not significantly related.

Table 5
Distribution of Subjects in each Caffeine Group by Caffeinism Status

	Daily Caffeine Intake			
	< 200mg	200-400mg	>400mg	Row Total
Caffeinism absent	68.4%	23.5%	8.1%	74.7%
(<5 Symptoms)	(n=93)	(n=32)	(n=11)	(n=136)
Caffeinism present	63.0%	26.1%	10.9%	25.3%
(≥ 5 Symptoms)	(n=29)	(n=12)	(n=5)	(n=46)
Column Total	67.0%	24.2%	8.8%	100%
	(n=122)	(n=44)	(n=16)	(n=182)

To explore these variables further, an analysis of the variance (using the Pillais statistic) of mean caffeinism scores ('CAFFSX11') across the three caffeine groups was conducted. The results indicated that the severity of caffeinism scores did not change significantly with increased caffeine consumption ('INTAKE') [F(2,184)=1.28, p=.192 (n.s)]. However, when the caffeine consumptions were not grouped (into high, moderate and low) but were entered as the initial values of 'TCAFF' (as seen in figure 5), ANOVA reached the .001 level of significance [F(10,184)=16.318, p<.001]. These results indicate that, on the basis of mean caffeine consumption, there is a significant difference between caffeinism scores.

Those who endorsed five or more signs of caffeinism ('INTOX') were compared to those who endorsed less than five symptoms to determine the significance of the various 'CAFFSX' questionnarie items. A multivariate analysis of variance of the 10 symptoms was significant [F(9,181) = 64.543, p<.001]. Discriminant analysis (univariate F-tests with (1,180) d.f.) indicate that those who were given the 'caffeinism present' label were more likely to report restlessness, nervousness, insomnia, diuresis, gastrointestinal disturbance, muscle twitching, impaired thought and speech, pounding heart, and periods of inexhaustibility (p<.001), and flushed face (p<.05).

Effect of caffeine consumption on performance-enhancing expectancies
The first hypothesis tested - that the extent of endorsement of personal expectancies about performance-enhancing properties of caffeine would be positively related to level of caffeine consumption and number of signs of caffeinism - was explored with the following results.

A stepwise multiple regression analysis was performed using the caffeine enhanced performance scale score ('EXPECT') and caffeine intake ('INTAKE') as independent variables, and the number of caffeinism symptoms ('CAFFSX11') as the dependent variable. Results of this analysis are shown in Table 6. 'EXPECT' and 'INTAKE' yielded a multiple R of .225 [$F(1,184) = 1.86$, $p=.163$ (n.s)]. The results of this test are not statistically significant indicating that there is no effect of expectancy score and caffeine consumption on the number of caffeinism symptoms endorsed by subjects. The null hypothesis can not be rejected at this low level of significance.

Table 6
Results of Stepwise Multiple Regression Using Number of Caffeinism Signs as the Dependent Variable

Variables	*Multiple R*	*R-squared*	*Beta*
EXPECT	.225	.051	.204
INTAKE			

The second hypothesis - that the syndrome present group would have significantly higher intake and more performance enhancement expectancies that the syndrome absent group - was tested statistically by use of a complex $\chi 2$ test comparing the 'EXPECT' score to caffeine intake 'INTAKE' and ANOVA comparing the effects of 'INTAKE' and 'INTOX' on 'EXPECT'.

The $\chi 2$ analysis of the relationship between the 'EXPECT' score (expectancy of CNS effects) and 'INTAKE' (caffeine consumption group) yielded a nonsignificant Pearson chi-square probability ($p=.855$). A multifactorial (3x2) test of significance was performed using the 'EXPECT' score as the dependent variable and 'INTAKE' and 'INTOX' as independent variables (Table 7). A significant F-ratio was revealed [$F(3,182)=3.84$, $p<.05$]. Most of the variance was attributable to 'INTOX' [$F(1,182)=8.66$, $p=.01$] rather than 'INTAKE' [$F(2,182)=1.08$, $p=.35$ (n.s)]. This indicates an association between caffeinism status and expectancy score, such that caffeinism status, but not the level of caffeine intake, can be used to predict expectancy scores (at least on the scale of caffeine's CNS stimulating effects). The null hypothesis can be rejected on the basis of these results.

Table 7
ANOVA: Change in EXPECT score with INTAKE and INTOX

Source of variation	*Sum of Squares*	*d/f*	*Mean Squares*	*F*	*Sig. of F*
Main effects:	27.110	3	9.037	3.841	.013
INTAKE	5.059	2	2.530	1.075	.347
INTOX	20.363	1	20.363	8.655	.004
2-way interaction:	1.658	2	.829	.352	.704
INTAKE INTOX					

ANOVAs assessing the effects of 'INTAKE' and 'INTOX' on 'POSITIVE', 'NEGATIVE', and 'ENHANCE' scores were systematically performed with the following results (Tables 8 to 10). Significant main effects were found for 'NEGATIVE' ($p<.05$) and 'ENHANCE' ($p<.01$) but not for 'POSITIVE' ($p=.22$). Most of the variance in the 'NEGATIVE' and 'ENHANCE' scores is attributable to caffeinism status ('INTOX') rather than caffeine intake. These statistics indicate that the 'NEGATIVE' expectancies scores and the 'ENHANCE' expectancies scores, respectively, are associated with caffeinism status, but not caffeine consumption. The 'POSITIVE' scale scores share no association with either 'INTAKE' or 'INTOX'.

Table 8
ANOVA: Change in POSITIVE score with INTAKE and INTOX

Source of variation	*Sum of Squares*	*d/f*	*Mean Squares*	*F*	*Sig. of F*
Main effects:	20.390	3	6.797	1.517	.221
INTAKE	7.136	2	3.568	.797	.456
INTOX	12.284	1	12.284	2.743	.104
2-way interaction	5.032	2	2.516	.562	.574
INTAKE INTOX					

Table 9
ANOVA: Change in NEGATIVE score with INTAKE and INTOX

Source of variation	*Sum of Squares*	*d/f*	*Mean Squares*	*F*	*Sig. of F*
Main effects:	63.712	3	21.237	3.207	.039
INTAKE	11.109	2	5.554	.839	.443
INTOX	53.363	1	53.363	8.058	.008
2-way interaction	.922	2	.461	.070	.933
INTAKE INTOX					

Table 10
ANOVA: Change in ENHANCE score with INTAKE and INTOX

Source of variation	*Sum of Squares*	*d/f*	*Mean Squares*	*F*	*Sig. of F*
Main effects:	124.946	3	41.649	8.031	.001
INTAKE	24.205	2	12.102	2.334	.118
INTOX	72.844	1	72.844	14.046	.001
2-way interaction	.169	1	.169	.033	.858
INTAKE INTOX					

A further multifactorial (3x2x2) ANOVA (Table 11) was conducted using the number of caffeinism symptoms ('CAFFSX11') as the dependent variable, and 'INTAKE' (high, moderate or low), 'CHOOSE' (the extent to which athletes purposely choose non-caffeinated beverages, rated as always or usually versus sometimes or never) and 'CAFUSER' (whether or not subjects use caffeine as a performance-enhancing drug in sport) as the independent variables. A significant main effect was obtained ($p<.001$), with 'INTAKE' being the main source of variation.

These results indicate that, on the basis of the number of caffeinism symptoms endorsed, there is a significant difference between those in each caffeine group [$F(2,183)=19.25, p<.001$], and between those who use caffeine as a performance enhancing drug versus those who do not [$F(1,183)=4.46, p<.05$], and between those who usually drink caffeinated beverages versus those who do not [$F(1,183)=4.16, p<.05$]. A number of two-way interactions (Table 11) are also significant.

Table 11
ANOVA: Change in CAFFSX11 with INTAKE, CHOOSE & CAFUSER

Source of variation	*Sum of Squares*	*d/f*	*Mean Squares*	*F*	*Sig. of F*
Main effects:	24.106	4	6.026	11.778	.000
INTAKE	19.698	2	9.849	19.250	.000
CHOOSE	2.126	1	2.126	4.156	.043
CAFUSER	2.282	1	2.282	4.460	.036
2-way interactions:	16.334	5	3.267	6.385	.000
INTAKE CHOOSE	2.153	2	1.077	2.104	.125
INTAKE CAFUSER	15.031	2	7.515	14.688	.000
CHOOSE CAFUSER	.021	1	.021	.042	.838

The extent to which athletes purposely choose ('CHOOSE') non-caffeinated beverages when selecting something to drink is as follows: 6 (3.2%) always, 25 (13.5%) usually, 70 (37.8%) sometimes, and 84 (45.4%) never. When asked whether their last drink was caffeinated or caffeine-free, 74 (40%) of the sample stated caffeinated, 105 (56.8%) stated caffeine-free, and 6 (3.2%) did not know.

Those who always or usually select non-caffeinated beverages (16.7%), were compared to those who rarely or never select caffeinated beverages (83.2%) to determine whether the two groups maintained different perceptions about the consequences of drinking caffeinated beverages. Multivariate analyses of variance on the 'EXPECT' scale score, the 'POSITIVE' scale score, the 'NEGATIVE' scale score, and the 'ENHANCE' scale score, all yielded non-significant F-ratios (p=.48, p=.95, p=.40, and p=.48 respectively). Discriminant analyses on individual scale items were therefore inconsequential. These results indicate that perceived psychoactive-, positive-, negative- and sports performance enhancing-effects are not strong indicators for selecting a beverage by those who prefer caffeinated beverages.

Regarding the 'PED' item, 26.5% of the sample believe caffeine enhances performance in their sport, 24.3% answered 'no', 13.5% believe it *sometimes* enhances performance, and 35.7% did not know whether caffeine is or is not a performance-enhancing drug.

When confronted with the question, "Do you consume caffeine for its performance enhancing effects?", 75.7% of the sample (n=140) claimed they had never used caffeine as a performance enhancing drug. The other 24.3% (n=45) admitted to using caffeine as an ergogenic aid. Of the 45 athletes who reported using caffeine as a performance enhancing drug, 7 (3.6%) had been using it for more than 5 years, 3 (1.6%) for between 4 and 5 years, 3 (1.6%) for between 3 and 4 years, 11 (5.9%) for between 2 and 3 years, 10 (5.4%) for between 1 and 2 years, and 12 (6.5%) athletes had been using it for less than one year. When asked whether they had ever intentionally used caffeine in competition knowing that they risked disqualification if a urine test showed high levels of caffeine, 17 (9.2%) of the entire sample responded 'Yes'. The sports with the greatest frequencies of caffeine 'users' were cycling and bodybuilding. Of the 12 bodybuilders using caffeine, only 2 had competed internationally, whereas all 13 of the cyclists who used caffeine as a PED were New Zealand representatives.

A number of ANOVAs were performed comparing athletes who used caffeine as a performance enhancing drug ('CAFUSER') with those who did not, to determine whether the two groups held different beliefs about caffeine. The scores of the groups on the respective expectancy scales were all significantly different ['EXPECT': $F(1,184)=22.148$, $p<.001$; 'POSITIVE': $F(1,184)=27.452$, $p<.001$; 'NEGATIVE': $F(1,184)=10.585$, $p<.05$; and 'ENHANCE': $F(1,184)=23.94$, $p<.001$]. These results indicate that athletes who use cafffeine as an ergogenic agent maintain different expectancies regarding caffeine's effects, than athletes who do not use caffeine as a performance enhancing drug. A further ANOVA revealed a non-significant difference between athletes who use caffeine as a PED and those who did not, on caffeine intake [$F(1,184)=.926$, $p=.398$ (n.s)].

A significant correlation coefficient was obtained for 'PED' (whether or not a subject believes caffeine is a performance enhancing drug) and 'ENHANCE' (the score on the scale that measures beliefs about the performance enhancing effects of caffeine in sport; $p<.001$). Similarly, significant correlation coefficients were reported for 'PED' and 'CAFUSER' ($p<.001$) and for 'CAFUSER' and 'ENHANCE' ($p<.001$). These results indicated that three significant relationships exist:

1. A relationship exists between athletes' beliefs in caffeine as a PED, and their expectations regarding caffeine-enhanced performance in sport;
2. An association exists between athletes' beliefs of caffeine as a PED, and their use of caffeine as a PED;
3. Athletes' use of caffeine as a PED and their expectancies regarding caffeine-enhanced performance in sport are related.

To corroborate this finding, an ANOVA was performed. A significant difference was obtained between those who believe caffeine is a 'PED' and those who do not, on their use of caffeine ('CAFUSER') as an ergogenic agent [$F(1,118)=40.600$, $p<.001$]. The effect of gender, as a co-variate in this analysis, was not significant ($p=.723$). This indicates that, on the basis of belief in caffeine as a PED (but not on the basis of gender), there is a significant difference between athletes who use caffeine as an ergogenic agent, and those who do not.

General Athlete Data In terms of the number of hours athletes spent training every week, 17.8% reported training more than 21 hours per week, 24.9% reported spending between 14 and 21 hours on their training, 34.1% spent between 7 and 14 hours training, and 23.2% spent less than 7 hours training per week. Over half of the sample (54.1%) had been involved in their sports for more than 6 years, 29.2% had been participating for between 3 and 6 years, and 16.8% had been active for less than 3 years. No further analyses on these data were undertaken.

Comparison of Athletic Identity and Sporting Achievement Descriptive statistics for the individual AIMS items are presented in Table 12. Subjects' scores (μ=26.33, σ=7.75) on the Athletic Identity Measurement Scale (Figure 7) were compared with their level of athletic participation (Table 13) with the following results. 51.4% of the sample identified strongly with the athlete role, scoring less than 25 on the AIMS. Corroboration with achievement data indicates that 48.6% had represented New Zealand and 30.3% competed at national level. Those whose highest level of participation was the regional level accounted for 13.0% of the sample, while 8.1% of respondents had only competed at local community level. The majority of participants who had a strong athletic identity had competed at national or international level.

Table 12
Descriptive Statistics for the AIMS Items

Item #	*Mean*[a]	*Std. Dev.*	*Item #*	*Mean*[a]	*Std. Dev.*
1	1.76	.97	*6*	2.57	1.16
2	1.86	.98	*7*	2.70	1.11
3	2.81	1.03	*8*	2.36	1.10
4	2.94	1.19	*9*	3.99	1.00
5	3.16	1.32	*10*	2.19	1.20

[a] Note. 1=strongly agree, 5=strongly disagree.

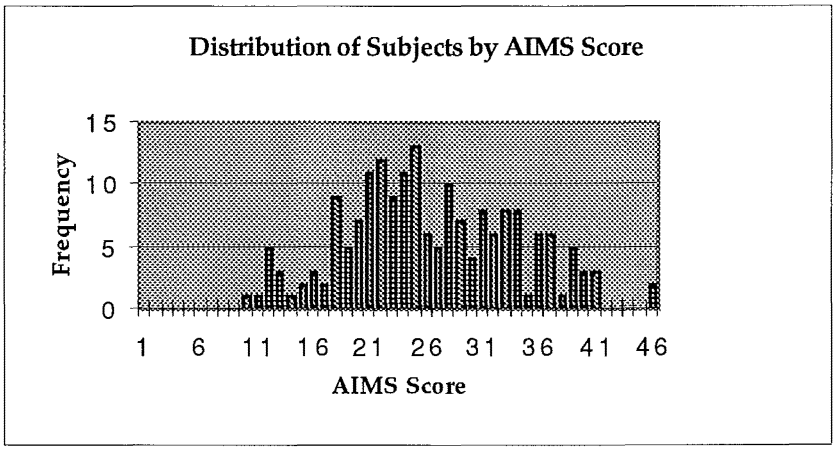


Figure 7 - Distribution of Subjects by Aims Score

Analysis of the variance between athlete's scores (Table 14) on the AIMS and their highest level of participation (Local/Regional/National or International) was significant [F(4, 183)=4.05, $p<.01$]. When levels of participation were combined (Local/Regional versus National/ International) the ANOVA result (Table 15) was also significant [F(1,182)=12.18,$p<.01$]. When gender was included in the analysis, it yielded a nonsignificant main effect [F(1,182)=1.36, $p=.24$]. When athletes were grouped differently (Local/Regional/National versus International) the result reached a higher level of significance [F(1, 182)=16.31, $p<.001$].

Table 13
Agreement between Athletic Identity & Level of Participation

Level of Participation	*High Athletic Identity (Score < 25)*		*Low Athletic Identity Score ≥ 25)*	
	n	%	n	%
Local Community	2	2.1	13	14.4
Regional	9	9.5	15	16.7
National	26	27.3	30	33.3
International	58	61.1	32	35.6
TOTALS	95	51.45% of the sample	90	48.6% of the sample

Table 14
ANOVA: Change in AIMS Score with GRADE and GENDER

Source of variation	*Sum of Squares*	*d/f*	*Mean Squares*	*F*	*Sig. of F*
Main effects:	3.624	4	.906	4.053	.004
GRADE	2.474	3	.825	3.690	.013
GENDER	.534	1	.534	2.388	.124
2-way interaction GRADE GENDER	2.409	3	.803	3.592	.015

Reliability and Construct Validity of the AIMS The reliability coefficient of the AIMS, using Cronbach's alpha, reached the .01 level of significance. Each of the reported ANOVAs provide support for the validity of AIMS. Most important for construct validity was the significant main effect for level of athletic involvement ('GRADE') on AIMS [$F(3, 182)=3.690$, $p<.05$]. As shown in Table 13, AIMS scores increased with level of athletic involvement. Further analyses comparing highest level of participation ('NATIONAL and INTERNATIONAL' versus others and 'NZREP' versus others) on the classification of strong athletic identity versus weak athletic identity (Tables 16 and 17 respectively) produced significant F-ratios. The main effect of gender on Athletic Identity was not statistically significant in either analysis [$F(1,182)=.090$, $p=.765$(n.s) and $F(1,182)=2.787$, $p=.097$(n.s) respectively].

Table 15
ANOVA: Change in AIMS Score by Elite and Gender (n=183)

Source of variation	*Sum of Squares*	*d/f*	*Mean Squares*	*F*	*Sig. of F*
Main Effects	798.874	2	399.437	8.196	.000
ELITE	593.642	1	593.642	12.180	.001
GENDER	66.476	1	66.476	1.364	.244
2-way Interaction ELITE GENDER	407.698	1	407.698	8.365	.004

Table 16
ANOVA: Identity Present or Absent by Gender and Elite (n=183)

Source of variation	*Sum of Squares*	*d/f*	*Mean Squares*	*F*	*Sig. of F*
Main Effects	2.158	2	1.079	4.731	.010
GENDER	.635	1	.635	2.782	.097
ELITE	1.021	1	1.021	4.478	.036
2-way Interactions	2.070	1	2.070	9.075	.003
GENDER ELITE					

Table 17
ANOVA: Identity Present or Absent and NZ REP (n=183)

Source of variation	*Sum of Squares*	*d/f*	*Mean Squares*	*F*	*Sig. of F*
Main Effects	2.070	2	1.035	4.447	.013
NZREP	2.069	1	2.069	8.888	.000
GENDER	.021	1	.021	.090	.765
2-way Interactions	.667	1	.667	2.864	.092
NZREP GENDER					

Table 18
Distribution of Subjects According to Gender and Level of Participation

New Zealand Representative		*GENDER*	
		FEMALE	MALE
n= 95	*NO*	n=67	n=28
n=90	*YES*	n=57	n=33

Subjects main 'MOTIVE' for participating in sport and their highest level of participation were compared (Table 19). Chi-square analysis of this relationship revealed a significant Pearson chi-square probability ($p<.001$) for 181 subjects. Four of the subjects had missing values and were not included in this analysis. The value of *phi* ($\phi=.539$) indicates rejection of the null hypothesis at .001 level of significance.

Table 19
Distribution of Subjects by Motive for Participation and Highest Level of Participation

MOTIVE	*GRADE*				
	Local Community	Regional	National	NZ Rep.	ROW TOTAL
Improve Skills	5	3	2	1	15 8.3%
Enjoy Competing	1	10	20	35	66 36.5%
Maintain Fitness	7	6	9	9	31 17.1%
Represent NZ		1	16	35	52 28.7%
Recognition	2		3	3	8 4.4%
Social Contact		2	4	2	8 4.4%
Enjoyment			1		1 .6%
COLUMN TOTAL	15 8.3%	22 12.2%	55 30.4%	89 49.2%	181 100%

The third hypothesis tested - that athletes identifying strongly with the athlete role would be more likely to use caffeine as a PED - was tested statistically by use of a 2x2 $\chi 2$ test comparing the athlete role and use of caffeine as an ergogenic drug [$\chi 2(1,184)=11.502$, $p<.001$]. Table 20 shows the cell frequencies in this equation. The result of this test indicates that the use of caffeine as a PED, and athletic identity are significantly related. The *phi* statistic for this relationship ($\phi=.249$) provides an indicator of the degree of relationship between the two variables. The null hypothesis can be rejected at this high level of significance.

Table 20
Athletic Identity by Caffeine 'User'

CAFFEINE USER	*STRONG ATHLETIC IDENTITY*	
	YES	NO
YES	37	8
NO	58	82

ANOVA revealed a significant difference between mean AIMS score and CAFUSER [F(1,183)=17.857, p<.001]. This can be taken to indicate that, on the basis of the AIMS scores (extent to which a subject identifies with the athlete role), there is a significant difference between those who use caffeine as an ergogenic agent and those who do not use caffeine in such a manner.

The final table to the results section (Table 21) shows the proportion of respondents in each of the twelve sports, the highest grade achieved, whether or not they believe caffeine is a PED, the extent to which they use caffeine as a PED in sport, and their caffeinism status.

Chi-square analysis of the relationship between athlete's sport and their use of caffeine as a PED yielded a significant Pearson chi-square probability (p<.001). Similarly, chi-square analysis of the relationship between athletes sport and their belief of caffeine's ability to enhance sport performance yielded a significant Pearson chi-square probability (p<.001). Clearly, these data warrant further investigation. Chi-square analysis of the relationship between sport and caffeinism status yielded a non-significant Pearson chi-square probability (p=.453).

Table 21
General Athlete Data

SPORT	*Grade*		*Believes caffeine is a PED*		*Uses caffeine as a PED in sport*		*Caffeinism status*	
	Up to National Level	New Zealand Rep	Yes	No	Yes	No	Present	Absent
athletics	6.3%	93.8%	62.5%	18.8%	25.0%	75.0%	6.3%	93.8%
(n=16)	(n=1)	(n=15)	(n=10)	(n=3)	(n=4)	(n=12)	(n=1)	(n=15)
boxing	100%		50%	50%	50%	50%	50%	50%
(n=2)	(n=2)		(n=1)	(n=1)	(n=1)	(n=1)	(n=1)	(n=1)
cycling	10%	90%	65.0%	20.0%	65.0%	35.0%	30.0%	70.0%
(n=20)	(n=2)	(n=18)	(n=13)	(n=4)	(n=13)	(n=7)	(n=6)	(n=14)
ice racing	46.2%	53.7%	7.7%	69.2%		100%	23.1%	76.9%
(n=13)	(n=6)	(n=7)	(n=1)	(n=9)		(n=13)	(n=3)	(n=10)
power lifting	80%	20%	33.3%	6.7%	27.0%	73.0%	40.0%	60.0%
(n=15)	(n=12)	(n=3)	(n=5)	(n=1)	(n=4)	(n=11)	(n=6)	(n=9)
rowing	63.2%	36.8%	36.8%	15.8%	21.1%	78.9%	31.6%	68.4%
(n=19)	(n=12)	(n=7)	(n=7)	(n=3)	(n=4)	(n=15)	(n=6)	(n=13)
shooting	33.3%	66.7%	33.3%	33.3%		100%	8.3%	83.3%
(n=12)	(n=4)	(n=8)	(n=4)	(n=4)		(n=12)	(n=1)	(n=10)
triathlon	77.8%	22.2%	66.7%	11.1%	22.2%	77.8%	22.2%	72.2%
(n=18)	(n=14)	(n=4)	(n=12)	(n=2)	(n=4)	(n=14)	(n=4)	(n=13)
wrestling	6.3%	93.8%	25.0%	31.3%	6.3%	93.8%	12.5%	87.5%
(n=16)	(n=1)	(n=15)	(n=4)	(n=5)	(n=1)	(n=15)	(n=2)	(n=14)
body building	88.9%	11.1%	77.8%	11.1%	66.7%	33.3%	33.3%	66.7%
(n=18)	(n=16)	(n=2)	(n=14)	(n=2)	(n=12)	(n=6)	(n=6)	(n=12)
fencing	47.6%	52.4%	14.3%	23.8%	9.5%	90.5%	33.3%	61.9%
(n=21)	(n=10)	(n=11)	(n=3)	(n=5)	(n=2)	(n=19)	(n=7)	(n=13)
archery	100%		6.7%	40.0%		100%	20.0%	80.0%
(n=15)	(n=15)		(n=1)	(n=6)		(n=15)	(n=3)	(n=12)
TOTALS	51.4%	48.6%	24.3%	75.7%	62.2%	37.8%	25.3%	74.7%
	(n=95)	(n=90)	(n=45)	(n=140)	(n=46)	(n=139)	(n=46)	(n=136)

CHAPTER SIX: DISCUSSION

Implications of the Results

Caffeinism Signs A substantial portion (25.3%) of this sample reported 5 or more 'caffeinism' signs. This finding, while clearly not a clinical appraisal of caffeinism, bolsters the importance of studying this syndrome in athlete samples where the ubiquitous use of caffeine is ingrained in the culture.

Caffeinism Severity and Caffeine Intake It has been suggested that quantity of caffeine consumed is strongly associated with caffeinism. This hypothesis implies that groups containing subjects with high levels of caffeinism should contain disproportionate numbers of high caffeine consumers. This hypotheses was tested on a sample of athletes. That the chi-square test comparing caffeine intake and caffeinism status was not significant, was surprising. Bradley and Petree (1990) revealed a significant association between the two. However, the divergent results between that and the present study could reflect methodological differences. Another possibility relates to individual sensitivity to the drug and a person's 'normal' intake. These possibilities are elaborated below.

Caffeine Expectancies The literature reviewed in chapters 1 and 2 indicates that caffeine has many physiological effects. Furthermore expectations play a part in the effects that caffeine exerts (Fillmore & Vogel-Sprott, 1992). The aim of the present study was to develop a scale (from findings of previous research) reflecting beliefs about caffeine's effects (Bradley & Petree, 1990; Page, 1987). Based on prior findings linking positive caffeine expectancies, high caffeine consumption, and negative caffeine-use outcomes, it was hypothesised that the extent of endorsement of personal expectancies of performance-enhancing properties of caffeine-containing beverages would be positively related to level of caffeine consumption and number of signs of caffeinism reported by each subject.

The results of this study do not suggest that expectancies regarding caffeine's CNS stimulating effects predict both increased caffeine consumption and problem outcomes (caffeinism). While the present study found an agreement between caffeinism status and expectancy scores, there was oddly no agreement between caffeinism status and caffeine intake. This may, in part, be due to individual sensitivity or habituation. For example,

those who do not usually drink caffeine, or who are sensitive to it, may experience 5 symptoms all at once when they do consume this drug (such as, insomnia, shaky hands, nervousness, gastrointestinal disturbance, and palpitations). These subjects can hardly be diagnosed with "caffeinism". By the same token, subjects who genuinely suffer from "caffeinism" may not experience the symptoms when they use caffeine, but may use caffeine to alleviate the *same* symptoms which may be brought about when an habitual consumer suddenly abstains (a withdrawal response).

The non-significant correlations observed between endorsement of performance-enhancement motives, caffeinism symptoms and caffeine consumption are not consistent with experimental findings that college students who drink coffee expect it will aid their psychomotor performance (Kirsch & Weixel, 1988). The questionnaire survey by Bradley and Petree (1990) also revealed an association between the use of caffeine for the 'common sense' purpose of exploiting its central nervous system stimulating effects ('EXPECT' motives) and reported levels of both caffeine intake and DSM-III caffeinism symptoms. Moreover, they found that syndrome-present subjects endorsed more expectancy items and consumed more caffeine than syndrome absent subjects. Again, the discrepancies between the studies could be due to the different subject pool (students versus athletes), research protocol differences, or methodological flaws in the present research.

The present study has revealed no association between expectancies of performance enhancement and caffeine intake. However, associations between the caffeinism status of subjects and their psychoactive, negative, and sporting enhancement expectancies were found. Whereas caffeine intake and caffeinism status were not good predictors of the expectancy score (involving caffeine's CNS stimulating effects), an athlete's use of caffeine as a PED in sport was a good predictor of the same expectancy score.

Furthermore, while CNS-stimulating expectancies were not a good predictor of the extent of caffeine consumption and the occurrence of caffeinism symptoms, the sport performance-enhancing expectancies measure was more useful in predicting caffeine consumption and caffeinism symptoms. The results also suggest that expectancies regarding sport performance enhancing effects ('ENHANCE' motives) are especially useful in predicting the use of caffeine as a performance enhancing drug in sport. These findings provide some support for the notion that sporting

expectancies can be valuable in predicting patterns of problem caffeine consumption. It must be noted that a liberty has being taken in describing caffeine consumption as problematic. For the purpose of this discussion the operational definition of problem caffeine consumption is met when the number of DSMIII-R "caffeinism" symptoms endorsed by a subject exceeds four. Caffeine consumption may also be seen as a problem by some people when it is being used (abused or misused?) for it performance-enhancing effects in sport.

In additional analyses, those who always or usually select non-caffeinated beverages were compared to those who rarely or never select caffeinated beverages to determine if they maintained different perceptions about the consequences of drinking caffeinated beverages. Multivariate analyses of the 10 positive statements and the 16 negative statements respectively, were non-significant. That is, the perceived and/or derived psychoactive effects (that is, gives one more energy, helps one to feel better, are relaxing) are not strong motivators for selecting a beverage by those who prefer caffeinated beverages. Similarly, those who usually select non-caffeinated beverages do not appear to refrain from caffeine-containing beverages because of the potential health implications and concerns, but for some, unknown, reason. These findings are in contrast to those of Page (1987) who found that subjects who rarely drink caffeinated beverages are more likely to have negative expectancies regarding caffeine's effects (that is, caffeine: is habit-forming; makes people more irritable, nervous, anxious and jittery; and, causes ulcers, headaches, kidney and bladder damage, high blood pressure and stomach upsets). Furthermore, Page (1987) found that people who do consume caffeinated beverages are more likely to perceive that these drinks give people more energy, help them relax, help them feel better, taste good, and are refreshing.

Despite, the results of the present study conflicting with Page's finding that the above perceived and/or derived effects of caffeine are strong motivators for either abstaining from or selecting a caffeinated beverage, the scale was still of some worth. That is, the positive effects listed above do appear to be strong motivators for those who use caffeine as a performance enhancing drug in sport. Even more significant is the finding that the perceived and/or derived sport performance enhancing effects of caffeine are strong motivators to using the drug during competition.

It is axiomatic that the findings of this study are not consistent with the behavioural expectancies which seem to have developed in our society. A cup of coffee is used to 'get going' in the morning. Over-the-counter caffeine tablets are sold to assist in combating fatigue, and decaffeinated coffee exists for those who experience nervousness or tension from caffeine. Virtually everyone has been exposed to information that would create the expectancy that caffeine is associated with symptoms such as reduction of fatigue or the production of nervousness.

Given the opportunity to develop these expectancies, it is interesting that the expectancy effect was found only on selected scales. Further discriminant analyses are required to reveal which individual items on these scales are associated with a stronger expectancy effect. In the interim, Appendix 20 and 21 give some indication of the particular weightings ascribed to each item on the EP.CAFF-1 and EP.CAFF-2 scales respectively. The Caffeinism Symptoms Questionnaire consisted of symptoms previously reported as being experienced by caffeine consumers which probably made it a more sensitive measure of the expectancy effect. Appendix 22 details the distribution of athletes by endorsement of caffeinism symptoms. It appears that diuresis and insomnia are the most common symptoms reported after consuming caffeinated beverages. However, conclusions can not yet be reached regarding which of the symptoms a significant expectancy effect exists for. It is likely that some symptoms are more susceptible to an expectancy effect than others.

Expectancies regarding caffeine's effects are very complicated and motivations for consuming caffeinated beverages are numerous. This may explain the discrepant findings between this and other research. For example, if a perceived negative such as a decrease in steadiness occurs on first exposure to caffeine, why do so many people continue to consume it? Perhaps the negative is outweighed by the concurrent effects which might be perceived as positive. This would be especially true is caffeine was initially sought for its expected and widely known alerting effect under conditions where a decrease in steadiness is not important, for example, in cycling but not in archery. The positive effects under those conditions combined with a quickly developing tolerance to the negatives might allow continued motivation for consumption.

On the other hand, initial consumption under conditions demanding fine motor control might tend to discourage further consumption. Other

work consistent with this hypothesis suggests that tolerance does not develop to the alerting effects of caffeine, but does develop to many other caffeine effects including jittering (Goldstein et al., 1969).

Loke (1988) reported that caffeine decreases boredom and relaxation and increases other ratings of subjective moods - anxiousness, tenseness, and nervousness. In addition to the alerting effects of caffeine, he suggests that real or expected mood changes experienced by regular caffeine users contribute to the motivation for consumption. Expected benefits for athletes might include improved performance on motor tasks, counteraction of fatigue-induced decrements, increased endurance, and enhancement of analgesic effects from over-the-counter pain relievers.

The behavioural effects of caffeine -for example, increased alertness and decreased fatigue - have generally been assumed to be due entirely to this drug. However, studies have revealed that variables such as personality and the time of day the caffeine is consumed also influence the experience of caffeine-related symptoms. Apparently, knowledge of having consumed a large dose of caffeine creates an expectancy of experiencing certain effects and this expectancy exacerbates the actual effect of caffeine. Indeed, the results of Christensen et al. (1980) revealed a significant expectancy effect on five caffeine-type responses (alertness, headache, rapid heart beat, sleepy, and clear flow of thought).

Because of the apparent complexities regarding cognitive functions, expectancies and the physiological effects of caffeine, overall conclusions about the effects of caffeine on physical performance are not possible at the present time. Research regarding the caffeine intakes and expectancies of athletes regarding this drug is in its infancy. Much needs to be done before firm conclusions can be drawn. Disparities in experimental methods have contributed to discrepant findings.

Athletic Identity The AIMS was found to be a reliable, internally consistent instrument. The validity data obtained in this study provide support for the claim that AIMS assesses identification with the athlete role. This replicates the findings of Brewer et al. (1993) who validated AIMS in a university population.

A major hypothesis tested by this study was that athletes who identify strongly with the athlete role are more likely to engage in drug-taking behaviour than athletes who have only a weak athletic identity. Furthermore, athletes who compete at a national or international level are more likely to have a strong athletic identity than those participating at local community or regional level.

This study showed a positive relationship between level of participation and athletic identity. Several potential risks for individuals with a strong athletic identity have been hypothesised (Brewer et al., 1993). For example, it is possible that a strong athletic identity may prompt individuals to engage in a sport or exercise activity to the extent that their physical health is jeopardised. In this study, the use of caffeine as a performance enhancing drug, was analysed as the sport activity that might jeopardise physical health. On the basis of athletic identity, there was a significant difference between those who use caffeine as an ergogenic agent and those who do not. In other words, athletes who 'use' caffeine as an ergogenic drug exhibit a stronger identification with the athlete role than non-users. The significant result can be taken to mean that caffeine use negates the potential health/fitness benefits of a strong athletic identity.

High levels of commitment commonly accompany participation in sport. Many individuals ascribe a great deal of psychological significance to their sporting involvement and identify with the athlete role (Brewer et al., 1993). These authors claimed that there may be both positive and negative consequences associated with strong athletic identity. On the one hand, committing oneself to the athlete role creates an opportunity to develop athletic skills, among other benefits. A strong, exclusive athletic identity may also have a positive effect on athletic performance.

Evaluation of the Present Study

The present study examined the role of expectancies and athletic identity on caffeine intake and caffeinism symptoms endorsed by subjects. However, it must be acknowledged that this study was not without limitations. The evaluation which follows addresses some of the general limitations of the study and specific methodological problems encountered.

Subject Selection The method of subject selection was less than perfect. This research was particularly constrained by practical aspects brought about by the Privacy Act. This meant that NSOs could not disclose athlete names to the researcher so subjects could not be contacted personally. Because athletes were contacted by their NSO or regional organisation, there is no guarantee that these respondents are a random sample or that they represent a cross-section of athletes. Furthermore, athletes could not be contacted personally to encourage the prompt return of questionnaires if they had not already been sent. This contributed to the smaller than optimum response. Time contraints meant that questionnaires could not be given directly to athletes by arranging visits to sports clubs around the Canterbury region or around New Zealand.

Follow up Data from a sample of non-respondents should have been obtained to establish the reasons why so many subjects took up their right for non-participation, and to determine that those who did not respond were not systematically different from those who had. This potential bias based on non-response could reflect both external and internal invalidity based on experimental mortality (that is, selective, non-random loss of subjects from the 'random' sample) as well as a potential increase in sampling error.

Expectancies Subjects were aware of the purpose of the study and were cued to possible effects of caffeine, which were described in the Perceived Consequences of Caffeine (EP-CAFF.1 & EP-CAFF.2) questionnaires. With questionnaire surveys there is always a danger that respondents will answer questions in ways that reflect what they believe the experimenter wants to hear ("pleasing the experimenter").

With regard to the AIMS scale, although athletic identity may be primarily a trait-like construct, AIMS scores may be susceptible to social and situational influences. Perhaps athletic identity is stronger when one is in the presence of athletes or in a sport environment than when one is in the presence of non-athletes or in a non-sport environment. These assumptions have implications for the completion of the questionnaire. A question arise, "Did athletes want to appear as if they identified with the athlete role?"

The Scale If a similar study is to be conducted, the researcher would do well to refine the questionnaire. For example, although the EP.CAFF-2 scale showed internal consistency, there were a number of items that could be re-worded. For example, the first four items opened with, "In my sport...." but the other items were not worded like that. While this wording allows the researcher to draw conclusions regarding the athletes' beliefs for their own sport, it does not expose an overall belief. For example, an archer may have a great deal of knowledge about caffeine, and knows that it can increase fat utilisation in some sports, but not in archery. This athlete would be forced to answer "No" to the item that contains the "In my sport..." prefix. However, if the item was re-worded, the response would be "Yes".

A second criticism of the scale is that the questionnaire involved many forced choice items. For example, "Caffeine helps people stay alert. Yes or No?" Such items could be redefined on a Likert scale to avoid the problems forced-choice scales bring to multivariate analysis (Barrett, 1995). Converting any forced-choice items to Likert configuration would render the scales less limiting. Five to 7 points along a continuum should provide the necessary distinctions to answer the questions, without providing respondents the opportunity to make finer distinctions than they are prepared to make.

Individual Questionnaire Items The questionnaire could have contained the question, "Do you deliberately refrain from consuming caffeinated beverages when competing/performing? Subjects were only asked if they did use caffeine as a performance enhancing drug. Furthermore, one respondent noted that they had intentionally used caffeine to enhance performance knowing the risk of disqualification but was not bothered by disqualification because "I have always been sure that I would not have been over the legal limit". These notes indicate that the questionnaire needs to be more comprehensive than its current form in order to widen its application.

Age Future research could do better by defining the age limits of athletes. The age cut-off could have been defined at 40 years. This present research showed a significant difference between age and caffeine intake. It is also likely that those in different age brackets have different expectancies regarding caffeine. Indeed, Swift & Tiplady (1988) found that caffeine produces changes predominantly in the direction of improved performance on attention tasks and choice-reaction time tasks for both young (18-37) and

elderly (65 - 75) subjects. However the same study showed that the elderly were less able than the young to report the acute subjective effects of caffeine such as increased drowsiness and dizziness. This has implications for the questionnaire requesting subjects to report the symptoms they experienced after caffeine consumption.

Habitual Caffeine Use and Tolerance to Symptoms Fisher et al. (1986) suggest that habitually high caffeine users acquire a tolerance to caffeine which reduces its effects during prolonged exercise and at rest. It seems that 4 or more days of withdrawal from caffeine resensitises an individual to caffeine's physiological effects. Because of the great individual variation in response to caffeine, subjects may differentially report caffeinism symptoms according to sensitivity to the drug, caffeine tolerance, and ingestion of large amount of caffeine. For example, a caffeine-sensitive individual may be classified as experiencing 'caffeine intoxication' even after 1 cup of coffee. This may be a reason why caffeinism and caffeine intake were not related in this study.

Individual Variation Caffeine makes some people more alert and energetic while it makes others feel nervous and shaky. This makes the ability to achieve statistical significance more difficult. The fact that this significance is not obtained, however, does not mean that for a number of subjects there is no ergogenic effect. Indeed, interpretation of nearly all reported studies is difficult since both positive and negative effects can be related to caffeine administration.

Averaging Caffeine Intake and Caffeinism Symptoms Caffeine research relies heavily on scale items to measure intake. Most research tends to collapse caffeine intake by creating averages (eg. low, moderate, high). Moreover, previous research has reported caffeinism according to whether it is present or absent rather than on a scale of severity. This project followed the trend. While measurements based on averaging techniques are valuable, the occurrence of any one or more symptoms at a severe level should be given special mathematical consideration to avoid being diluted beyond recognition in the averaging process.

The same criticsm applies to caffeine intake. For example, high intake was considered to be above 400mg per day in this study. This ignores the fact that a number of respondents consumed much more, in several cases, over 800mg per day, and in one case, over 1000mg per day. That a significant

relationship was found between the 11 caffeine intake groups and the number of caffeinism signs (ranging from 1 to 10), but not between the 3 broader caffeine intake groups and caffeinism status (less than 5 signs versus 5 or more signs), is an excellent example of how the 'power' of a finding can be drastically reduced by regrouping data into a smaller (perhaps less meaningful) number of categories.

On this note, data analyses would have been easier and more significant if real means could be calculated (for example, on the age and income items). That is, categorical and nominal variables can sometimes be difficult to work with. It also means a lot of manipulation is required when doing data analyses to recode the variables into more workable data.

Response Patterns In this study, indications of caffeinism did not vary markedly from subject to subject (Appendix22). Some scale items seemed very relevant (for example, diuresis and insomnia) while others were of little value (for example, flushed face).

Sampling Problem One of the reasons for the unsatisfactory results of this study may be the absence of an adequate consideration that it is specific. The lack of such an approach appears in the selection of the athletes. The study of athletes from various sports, combined in a heterogenous group, did not permit discovery of the personality characteristics which are different for the various sports. Also, this survey should be undertaken within a research design using comparison groups. That is, the athletes were not compared with non-athletes. Research on the athletic identity and personality of athletes needs simultaneous comparisons in two directions: athletes from separate sports with non-athletes and athletes of various sports among them. Socio-demographical factors have a great impact on personality and the specific sport an athlete choses (Geron et al., 1986) and should have been considered in this research.

The personality profiles of athletes can not be considered as absolutes. They always appear relative to the population to which the athletes are compared. Participants in a sport differ from the non-athletic population by certain features and from the athletes of other sports, by other features. Therefore, when making the assumption that athletes scoring high on AIMS would be more likely to use caffeine as a PED; the sport engaged in and personality profile of the athletes, should have been taken into account.

Statistical Manipulation of the Scales Clearly, a more detailed data analysis would provide more comprehensive answers to the research questions. Additional suggested analyses include factor analyses of the individual items of the expectancy scales, the CAFFSX questionnaire, and the AIMS questionnaire. This would provide an indication of the extent to which each item contributes to the final scale score.

Differences in Knowledge Subjects were asked whether the last drink they had was caffeinated or not. Most knew although a number were not sure. Respondent #236 did not know that Diet Coke contained caffeine, respondent # 333 did not know that water contained no caffeine, and respondent # 308 did not know that apple juice was non-caffeinated. A questionnaire more sensitive to these differences in subject knowledge regarding caffeine needs to be devised.

Participant Criticism of the Scale Feedback in the form of handwritten margin notes on the questionnaire or separate letters from participants in the study raises three points for consideration. One participant (a target shooter) found the questionnaire limiting with regard that sport, in that it was not sufficiently detailed. Another (an archer) noted that caffeine exacerbated her arthritis symptoms. As a result she rarely consumed caffeinated beverages, and therefore believed she was in no position to complete the caffeinism symptoms questionnaire. The third issue relates to the variability of subject responses and shows that forced choice questionnaires, are by definition, limiting (as discussed above). I take the liberty of demonstrating this point with the notes of two respondents, the first an archer, and the second, a fencer:

> "In fairness I should point out that in my medical notes my doctor has branded me as a "caffeine addict". The quantities I indicated are averages, I can drink up to 16 cups a day, and have for the past 13-14 years. I use it to wake up, sleep, relax and wind-up. I carry on this pattern while I'm participating in sport, not for enhancement, but for survival. I can get into the position that if I don't have it I get headaches and sometimes, although not often, three or four cups in quick succession will give me a headache. I'm finding recently that I'm getting the shakes if I haven't had coffee so I drink a glass of coke for quick relief." (anon.)
>
> "...caffeine is very bad for my performance in fencing. If anything I require relaxing before competition." (anon.)

Suggestions for Future Research

It is an additional methodological criticism of the present study that leads to suggestions for future research: the methodology used in this study 'wastes' data. That is, there are many data generated from this project that were not used. A number of research ideas, which can be answered by the analysis of this data and the generation of further data, merit exploration. The ideas fall into four categories - issues involving the personality of athletes, the use of other drugs, sport specific expectancies, and motives and rationalisations for caffeine use in sport.

Personality of Athletes This study made the assumption that athletes with a strong athletic identity would be more likely (than athletes who do not identify strongly with the athlete role) to engage in a sport activity to the extent that their physical health is jeopardised. The exercise activity in question was caffeine abuse. However, there are two flaws in this assumption.

First, caffeine consumption can not be regarded as a dangerous behaviour relative to other behaviours an athlete might engage in, such as competing with a serious injury or the use of anabolic steroids. Indeed, caffeine's ubiquity places it in a position that it is rarely classified as a drug by the non-scientific community. Therefore, illicit and arguably more dangerous drugs, such as anabolic steroids may be more appropriate when comparing behaviour with AIMS.

Second, the personality profile of an athlete may have a bearing on the propensity to use performance-enhancing drugs. Research suggests that the personality profiles of athletes in various sports are not homogeneous (Geron et al., 1986). Although some consistent personality characteristics have been found for athletes, there are also some differences. For example, sprinters have been found to be more anxious, emotionally disturbed and tense than a matched sample and athletes of other sports. These personality characteristics, which are generally accepted as unfavourable, may be favourable for participation in this sport, thus justifying that high level athletes possess them. It is worth investigating whether athletes of different sports exert personality characteristics indicative of drug taking behaviours.

Another application of caffeine research would be to examine the relationship between caffeine consumption and the incidence of caffeinism in athletes using the State-Trait Anxiety Inventory (STAI) and the Beck Depression Scale (BDE). This would provide a further indication of the relationship between personality (specifically trait anxiety and depression) and use of caffeine as a performance enhancing drug. It may be that the STAI and the BDE are more reliable indicators of problem caffeine use than the CAFFSX scale developed for this study.

Utility of the AIMS A great deal of attention has been focused on the relationship between personality and sports participation. Definitive conclusions regarding the presence of an 'athletic personality' and the consequences of various levels of athletic participation have been limited by methodological shortcomings and equivocal findings (Hauck and Blumenthal, 1992). An interesting comparison could be made of athletes scores on the Minnesota Multiphasic Personality Inventory and AIMS.

Another obvious use for the AIMS is to investigate the relationship of athletic identity to drug taking by athletes. It seems that this scale is sensitive to caffeine, despite its ubiquitous nature. The association of AIMS to the use of other drugs could also be tested. Should a strong, exclusive athletic identity be found in subsequent research to function as a vulnerability factor for drug-taking, the AIMS could be used as a means of identifying individuals who are at risk. Preventive interventions could then be implemented to help athletes at risk avoid drugs. Perhaps interventions involving valuing competence in non-sport activities would prevent athletes from placing too much importance on sporting success and from resorting to drug-taking to achieve that success.

In this study, males and females did not score differently on the AIMS. Brewer et al. (1993) revealed a significant difference on the basis of gender on AIMS scores, suggesting a greater emphasis in United States' society on sport for males than for females. That the interaction between sex and level of athletic involvement was no statistically significant in this study does not necessarily mean that an equal emphasis is placed on sport for both males and females in New Zealand. Rather it indicates that the two studies tested different subject pools (college students and athletes). If this study was to be replicated and AIMS tested using a matched sample of New Zealand university students, males may well score higher than female on AIMS.

Rationalisations for Caffeine Use and Sport Participation Motives It would be valuable to obtain athlete's attitudes and rationalisations for caffeine use as a PED: why do athletes use caffeine, where did they learn about the effects of caffeine, and what values do they associate with this form of drug use? For example, if athletes are using caffeine as a means of reducing body fat, are they also exhibiting any of the symptoms of anorexia or bulimia nervosa? In this study assumptions were made about athletes' motivations for caffeine use based on their sport. It was assumed that the motives for using caffeine use during competition would largely be related to decreased fatigue and increased time to exhaustion, to increased alertness and vigilance, and to weight reduction. However, athletes may well use caffeine for effects not related to performance enhancement. Such reasons are: the apparent reduction of negative psychological reaction to challenge; relaxation; to buffer withdrawal symptoms experienced after a period of abstinence; or, as a normal part of their socialising.

It is imperative to recognise athletes' reasons for taking caffeine so that strategies can be devised to reduce, if not eliminate, the problem (if it can be considered a problem). This is especially so if athletes are displaying symptoms of caffeinism. Younger athletes are particularly susceptible to the health hazards of caffeine. Research into understanding the causes of caffeine use among athletes remains scant, perhaps because it is a recreational drug of a ubiquitous nature. However, it is also a potential performance enhancer and a potential health hazard. In order to educate we need know whether caffeine is used for the need to be competitive, to increase strength, to cope with stress, to reduce pain, to relax, to control weight, or to overcome boredom.

Another useful application of this research would be to study the extent to which athletes sport participation motives (Ryckman & Hamel, 1993) impact on their AIMS and their use of caffeine. For example, future research could examine the importance of intrinsic versus extrinsic reasons for the participation in sports, and their relationship to athletes' AIMS and caffeine intakes. Participation motives that could be studied include: improvement of skills, enjoyment of competition, maintenance of fitness, wish to represent New Zealand, recognition, social contact. While this information was elicited from subjects in the present study, no comparisons were made. However, interesting and valuable information could be provided about the particular motives athletes scoring higher on AIMS consider important reasons for participation in sport.

Other Drugs Two considerations are important here. First, because of the ubiquitous nature of caffeine, this drug is not an easy one to study in relation to sport. It is apparent that many athletes, as yet, do not have the relevant information available to them regarding caffeine and its potential as an ergogenic agent. Therefore, other ergogenic drugs such as amphetamines or steroids may have been easier to study. Furthermore, it may be interesting to examine whether athletes using caffeine as a performance enhancing drug are also using illicit drugs on the sports field.

Future research could also examine subjects use of other social drugs such as nicotine and alcohol, their interactions with caffeine, and athletes perceptions thereof. For example, Kerv et al. (1991) showed that motor function was facilitated by nicotine and caffeine and that the debilitating effects of alcohol were frequently antagonised by either drug. Others have found that with the exception of reaction time tests, caffeine does not antagonise ethanol-induce performance decrements (Millis, 1987). Combinations of these social drugs need to be considered. However one would assume that athletes would be non-smokers and abstainers from alcohol or at the very least, only light drinkers and smokers.

Expectancies and Sport Future research could do well to assess which sport-specific expectancies athletes of various sports maintain. For example, does an archer or shooter know more about caffeine's effects on anxiety and tremor than on the sparing of muscle glycogen? Would this be reversed for cyclists and triathletes? The data was available to answer such questions but the additional analyses required were not undertaken at the time of writing. Discriminant analyses could be conducted to determine if there are any items on the EP.CAFF-2 scale which stand out for particular sports.

Some researchers (Butts & Crowell, 1985; Flinn et al., 1990) suggest that only well-trained athletes can benefit from caffeine. However, Casal and Leon (1982) have suggested that caffeine only has a positive effect on non-elite athletes since they do not have the increased lipolytic enzyme activity, mitochondrial density or size that endurance activities brings about. Do these athletes perceive caffeine's effects differently? Similarly athletes with high amounts of caffeine in their normal diet have been found to build up a tolerance to the substance (Fisher et al., 1986) which prevents caffeine from having its purported ergogenic effect. Does this influence performance enhancing expectancies? Obviously, there is a great deal of scope remaining for the investigation of caffeine in sport.

Educating Our Athletes

One of the functions of the NZSDA is to "foster the development of educational programmes and materials which will reduce the unethical use of drugs and doping methods in sport" (Hatherton, 1991, p13). The same report notes that the "correct use of the sport sciences should be promoted and developed as an alternative to misuse of PEDs" (p15). However, estimates of the cost of an education programme involving specific drugs-in-sport educational material and methods stand at around $250 000 per annum. When this amount of money is concerned, it is not surprising that drug education efforts are focused on the drugs with more 'serious' effects than caffeine, such as the anabolic steroids, narcotics, and amphetamines.

Drug education is an important facet of school programmes today. The Foundation for Alcohol an Drug Education (FADE) has been set up to highlight the risks of alcohol and drug abuse to school children. Popular athletes are spokespeople for the foundation. Caffeine is not on the programme and because of the ubiquitous nature of caffeine in society the "Scared Straight" type of education does not seem relevant.

The question arises, should our athletes and our youth be educated about the potential effects of caffeine. Given the limited budget and the necessity for education about other drugs, caffeine education is not a priority. Furthermore, caffeine is only banned in high doses so perhaps athletes could be educated as to how caffeine can be used safely and legally to enhance their sporting performance. Thus caffeine could be used to its full ergogenic effects as a nutritive aid whilst the athlete's health is not compromised, and nor is she or he cheating.

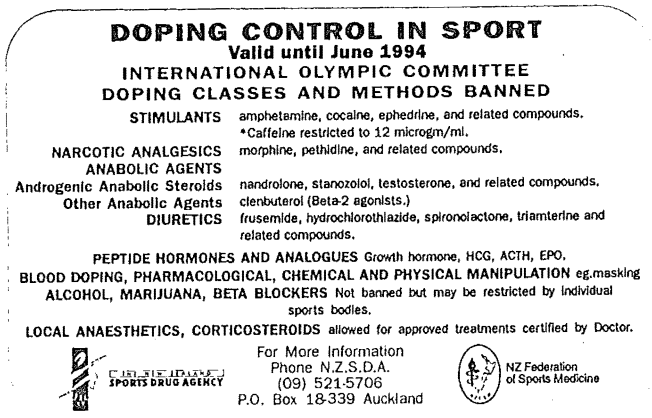
Despite caffeine being a relatively safe drug, there is a place for education about its effects. There can be little doubt that many adolescent athletes are taking caffeine. As a result, they are also experiencing the toxic effects of this drug - tremor, hyperactivity and diuresis. Although cola beans themselves contain little caffeine, the drug is intentionally added by soft drink manufacturers, making cola drinks the major source of caffeine for adolescents even if they are unaware they are consuming a drug.

Athletes need to be alerted to the fact that caffeine is a drug and that caffeine-containing beverages do have the potential to influence mood. Furthermore, the psychoactivity of caffeinated beverages creates some

possibility of dependence. Educational efforts should also include information about which food and drink products contain caffeine and the respective amounts of caffeine they contain. Accurate and current information about the potential health effects of caffeine consumption should also be provided to athletes.

The First Permanent World Conference on Doping in Sport (1988; cited in Hatherton, 1991) considers three elements to anti-doping education. The first involves educating athletes and significant others about the ethical basis of sport, and the notion that doping is 'cheating'. The second involves educating athletes of the dangers of drugs from a health standpoint. The third involves providing information about national and international anti-doping rules and the lists of classes of banned substances.

Regarding the first consideration, this element of education does not have to be made specific to the different classes of drugs. Therefore, this element of education is already covered for caffeine. Concerning the second point, that athletes need to be educated about the negative health consequences of doping. Research suggests that caffeine can be a relatively safe drug, so it might not be necessary to educate athletes on this level (except if they are abusing very large quantities). On the third point, New Zealand's top athletes are provided with the information they need, even for caffeine. They are issued with a plastic wallet-sized quick reference card. One side (Figure 8) gives a list of doping classes and methods banned, while the reverse side (not shown) provides examples of permitted and banned substances for various symptoms (for example, asthma, cough, headache). Such information is not given automatically to lower grade athletes, but it is available upon request. Whether the current education philosophy for caffeine is adequate or not, remains to be seen.



DOPING CONTROL IN SPORT
Valid until June 1994
INTERNATIONAL OLYMPIC COMMITTEE
DOPING CLASSES AND METHODS BANNED

STIMULANTS	amphetamine, cocaine, ephedrine, and related compounds. *Caffeine restricted to 12 microgm/ml.
NARCOTIC ANALGESICS	morphine, pethidine, and related compounds.
ANABOLIC AGENTS	
Androgenic Anabolic Steroids	nandrolone, stanozolol, testosterone, and related compounds.
Other Anabolic Agents	clenbuterol (Beta-2 agonists.)
DIURETICS	frusemide, hydrochlorothiazide, spironolactone, triamterine and related compounds.

PEPTIDE HORMONES AND ANALOGUES Growth hormone, HCG, ACTH, EPO.
BLOOD DOPING, PHARMACOLOGICAL, CHEMICAL AND PHYSICAL MANIPULATION eg.masking
ALCOHOL, MARIJUANA, BETA BLOCKERS Not banned but may be restricted by individual sports bodies.
LOCAL ANAESTHETICS, CORTICOSTEROIDS allowed for approved treatments certified by Doctor.

SPORTS DRUG AGENCY

For More Information
Phone N.Z.S.D.A.
(09) 521-5706
P.O. Box 18-339 Auckland

NZ Federation of Sports Medicine

Figure 8 - NZSDA Card given to Elite Athletes. This card provides summary information of doping controls in sport.

Implications for Athletes

Athletes have used caffeine as an ergogenic aid for many years. The exact mechanism of that aid is unclear because of caffeine's broad performance-altering metabolic and physiological effects. The following discussion is intended to provide athletes, their coaches and influential others, with valuable, objective information regarding caffeine's place in sport.

Doping and the International Olympic Committee The IOC has banned 'high levels' of caffeine, but monitoring its use and abuse is difficult. Caffeine is a legal drug and athletes are free to experiment with its use. However, enhanced performance, as evidenced by continuously improved records since the turn of the century, correlates much more with improved training methods, better materials and equipment, improved nutrition, and improved psychological techniques than with the use of ergogenic drugs. The IOC considers caffeine to be a drug and does not sanction its use by athletes. The pertinent research has shown some positive implications for caffeine's performance enhancing effects, but the use of caffeine by athletes as an ergogenic aid cannot be condoned.

Caffeine fits into three categories. It can be used to diminish the perception of pain and therefore reduce suffering from injury as an end in itself (therapeutic use of the drug) rather than as a means to an end (improving performance or altering perceptions). Thus the explanations and categories for drug use may not always be mutually exclusive. Rather, they are interactive. Since caffeine affects most systems of the body, athletes who wish to experiment with it should do so before competing in any world-class or critical event.

Sport Performance Variables Caffeine affects muscle function, and it has been shown to enhance performance in endurance events and in activities requiring increased alertness, decreased reaction time, and lowered anxiety levels, such as archery and shooting. Studies have shown that caffeine has no ergogenic effect on large-muscle, short-term intense exercise requiring strength and power (Van Handel, 1983).

The substrates that are used as fuel for endurance activities are mainly free fatty acids and muscle glycogen. The longer muscle glycogen is spared, the greater the delay in the onset of exhaustion (Ivy et al., 1979). Caffeine's

ability to mobilise plasma free fatty acids is theorised to have a glycogen-sparing action during exercise (Slavin & Joensen, 1985). According to the American Dietetic Association, the theorised glycogen-sparing action of caffeine may offset fatigue, but its negative side effects may outweigh the possible benefits (Smith, 1987).

Contradictory data abound with respect to caffeine's effect on other measures of performance that are important in athletic competition. Poorly substantiated reports of less drowsiness, increased vigilance, reduced fatigue, and an increased capacity for sustained concentration have all contributed to the widespread use of caffeine (Powers & Dodd, 1985). Subjective feelings of increased alertness and nervousness are evident when no objective improvement in alertness, psychomotor performance following caffeine ingestion exist (Goldstein et al., 1965).

The Magic Dose The positive effects of caffeine appear to be dose-related. However, the actual amount of caffeine needed to enhance performance is still not known and individual variability will affect the amount. The literature suggests that an intake of 4 to 5 mg per kilogram of body weight (2 cups of coffee [300mg] for a 70kg person) results in a significant plasma caffeine level within 15 minutes (Essig et al., 1980). Furthermore, the positive effects of caffeine also to be affected by the amount of caffeine normally consumed by the subject.

Masking of Fatigue Caffeine can produce a physiological masking of fatigue (Lombardo, 1986) which causes athletes to be unaware of changes to various physiological parameters, even when they reach dangerous levels. That is, instead of preventing fatigue (although it may delay it through glycogen sparing) caffeine masks the effects of fatigue and interferes with the body's fatigue-alarm system, which could be disastrous under extreme environmental conditions (Dyment, 1987). That caffeine can allow athletes to continue to exercise at high levels of effort for a longer period and endure a higher level of anaerobic metabolism poses a threat. In short-distance events this may not be dangerous but in events lasting for more than an hour, the failure to be aware of 'danger signals' and to react to them could be an threat to life.

Anxiety Shanahan & Hughes (1986) have found that caffeine can potentiate performance-induced anxiety. This has implications in the sports arena. Athletes will have to weigh up this affect of caffeine and the reported

benefits. If caffeine use is viewed as a means to ameliorate the emotional effects of competitive stress, caffeine seems an unlikely drug to use for that purpose. This being the case, caffeine consumption should be decreased during sport, especially if an athlete is particularly vulnerable to stress.

A high caffeine intake is has also been associated with symptoms of anxiety neurosis. However, Raitliff-Crain et al. (1989) found that caffeine can reduce negative mood and discomfort attributed to challenge while increasing indices of sympathetic arousal. The stimulating properties of coffee, coupled with its apparent buffeting of perceived stress places it in a paradoxical position.

Habitual Caffeine Use Although positive effects of caffeine administration may occur for parameters associated with general perception of well-being or reaction time, it is perhaps equally likely that negative effects may occur depending upon the current caffeine status of the subject. It may also be true that administration of caffeine after a period of forced abstinence may be of benefit to an athlete considered a regular user. In other words, athletes who abstain from caffeine for several days and then resume intake apparently receive better results than chronic users.

Definitive tests have shown that habitual caffeine users establish a tolerance to caffeine that is evident both at rest and during exercise (Fisher et al., 1986). That is, regular consumption diminishes its stimulant effects so that increasing dosages are needed to get that effect. However, the occasional user of caffeine will probably notice increased effects from smaller doses of the drug. When tolerance to coffee does occur, resensitisation to caffeine's physiological effects can occur by increasing the dose, or by complete abstinence over as little as four days.

Bullet Point Summary of Caffeine's Effects

- Physical dependence on caffeine can develop and withdrawal symptoms may occur upon cessation of caffeine (Holtzman, 1990).
- Athletes who habitually consume caffeine need to abstain from caffeine for 4 days in order to magnify its effects.
- High intakes may result in fluid loss, polyuria, increased heart rate, and an increased anxiety level that may outweigh any positive effects on fuel utilisation or alertness.
- Well-trained endurance athletes already use fatty acids effectively and thus spare their glycogen. The benefits of caffeine may therefore be small.
- Carbohydrate loading negates the ergogenic effect of caffeine by dampening its fat mobilising effect.
- Caffeine-induced improvements in aerobic exercise (incremental or steady state) are related to increases in free fatty acid utilisation, thereby sparing carbohydrate and delaying the onset of fatigue.
- Caffeine needs to be taken 3 to 4 hours before aerobic exercise in order for FFA levels to reach their peak to take advantage of the glycogen-sparing effects.
- Anaerobic exercise tasks should commence 1 hour after caffeine ingestion when plasma levels are highest.
- Caffeine-induced increases in anaerobic exercise may be more likely due to neurologic stimulation, or a reduction in the sensation of fatigue, but not to use of fuel.
- Due to alterations in fluid and electrolyte balance, caffeine ingestion has potential deleterious effects on work tolerance and thermal stress.
- Impaired memory, pronounced anxiety, and sensory disturbances (hallucinations) are some of the results of ingesting massive doses of caffeine by humans under prolonged competitive stress (Stillner, 1978).
- The benefits sought from caffeine's ergogenic properties (eg. delay in the onset of fatigue, weight control) must be weighed against any potential cost (eg. decreased accuracy, impaired muscular coordination, diuresis).
- Individuals engaged in sensitive and hand steadiness-dependent sports could benefit form the knowledge that a reduction of caffeine intake may avoid tremor and loss of fine motor coordination.
- Athletes should consider previous caffeine tolerance and their body size before estimating appropriate caffeine dosage. It would be prudent to keep caffeine consumption to within 2 to 3 cups of coffee (5mg.kg-1) prior to sports activity (Slavin & Joensen, 1985).
- The use of caffeine in sport should be viewed with caution. It can cause adverse effects as mentioned previously. In addition, there may be individuals who are particularly sensitive to caffeine, resulting in over-stimulation of body systems and thus causing the opposite effect in performance from that desired.
- Watson (1988) gives practical solutions to those wishing to reduce or eliminate their caffeine consumption.

Negative Consequences of Caffeine Use for Athletes

Possible side effects that could limit sports performance include insomnia, withdrawal headache, diarrhoea, anxiety, irritability, and increased urine production. Individual response to caffeine differs and depends on past use, body composition, and dosage.

From an athletic perspective, high intakes of caffeine may result in fluid loss, polyuria, nausea, increased heart rate and anxiety. These effects may outweigh the positive effects of fuel mobilisation and alertness. Even modest doses cause side effects of tremor, insomnia hyperactivity and a diuretic effect with possible diarrhoea (Passmore, 1987). However, the health consequences of acute and chronic caffeine intake appear to be minimal compared with other stimulants. Moreover, few restrictions are placed on the use of caffeine. These considerations suggest that caffeine can indeed be a convenient, relatively safe and useful drug for athletes. A number of health issues of concern to athletes do warrant discussion. These are athletic amenorrhoea, eating disorders, diuretic tendencies, thermo-regulation, food iron absorption, and toxicity of caffeine.

Athletic Amenorrhoea This is a major problem affecting women participating in competitive sports (Hellemans, 1990). Concern is related to the low bone density levels of many of these women which predisposes them to osteoporotic fractures. Diet is implicated in the aetiology of athletic amenorrhoea (Myerson et al., 1991), including an association with a low calcium diet. Ingesting excessive caffeine increases calcium excretion about 50% (Massey & Hollingbery, 1988), meaning even less calcium is available for bone health (Shah & Belonje, 1988). High caffeine consumption has been proposed as a risk factor for osteoporotic fracture, but the evidence associating high caffeine intake with low bone density is inconsistent (Cooper et al., 1992).

Eating Disorders These are also prevalent among female athletes. Male athletes may also suffer from eating disorders, especially those who have to 'make weight'. Caffeine abuse has been linked to bulimia and anorexia nervosa (Fahy & Treasure, 1991; Pruitt et al., 1991). Patients use caffeine for its appetite suppressant, diuretic, and stimulant effects, and are likely to suffer from caffeine intoxication. Indeed, caffeine has been used in weight loss preparations and regimens. This is a result of its negligible energy values and its thermogenic properties (Astrup et al., 1990; Curatolo &

Robertson, 1983; Dulloo et al., 1989). Caffeine increases the metabolic rate during the first several hours after ingestion by 14% in non-users and occasional users, but not in habitual users (Poehlman et al., 1988). Endurance-trained athletes also have a blunted response to the thermogenic actions of caffeine (Poehlman et al., 1988). Wilcox (1982) demonstrated that fat loss by rats doing aerobic exercise can be increased when caffeine is ingested prior to training sessions. Body weight, fat-pad weight and fat-cell size were all significantly lower in the caffeine trials. Some athletes take caffeine for its supposed weight loss benefits. One mechanism for this effect is the short-term reduction of calorie intake following caffeine consumption, but this effect seems to be restricted to males (Tremblay et al., 1988).

For men using caffeine, care should be taken to ensure their dietary needs are met, especially their protein intake. The mean caloric reduction following caffeine ingestion was 21.7% less than under placebo conditions (Tremblay et al., 1988). This reduction could have deleterious effects on athletic performance, especially if the deficit continues over a period of time, and lean body mass is compromised. Energy expenditure increases with caffeine in men but not women, that is, a greater disturbance of homeostasis occurs in men than women, when factors affecting the sympathetic nervous system, such as drugs and exercise are studied.

Diuretic Properties and Urinary Mineral Excretion Caffeine has long been touted as a diuretic (Bellet et al., 1969), increasing an athlete's risk for dehydration and heat-related illness when performing in hot environments. Hellemens (1991) and Clement (1991) discuss the importance of adequate hydration for both athletic performance and regulation of body temperature. Some authors report that caffeine does not cause a significant diuretic effect (Birkett et al., 1990; Massey & Hollingbery, 1988). However, Passmore et al. (1987) found that caffeine ingestion increases urine volume as well as sodium, chloride and potassium excretion. A balance of electrolytes is particularly important during training and competition. In comparison to other diuretics, caffeine is one of relatively low potency, and tolerance to this effect does develop (Curatolo & Robertson, 1983). Caffeine also acts on the smooth muscle (Graham, 1978) of the gastrointestinal tract, suppressing motility in the stomach and small intestine, but relaxing the muscle of the large intestine. An endurance athlete should think twice about ingesting caffeine before an event, during which even a minor case of diarrhoea could be a major inconvenience!

Body Fluid Balance and Thermo-regulation Normally, exercise- and/or thermally-induced sweating reduces urine output, thus conserving both water and electrolytes. A large caffeine-induced diuresis under these conditions may compromise the circulatory and sweat response to internal heat production. However, the effect of caffeine on body temperature is difficult to interpret. It may be a consequence of increased motility, a calorigenic effect, or a direct central effect on temperature regulation. Waldeck (1973) found that caffeine partially counteracted the hypothermia caused by reserpine in mice. Falk et al. (1989) found that caffeine ($2.5mg.kg^{-1}$) did not produce any significant adverse effects on body fluid balance or on the thermoregulatory and metabolic response in subjects exercising in thermoneutral conditions. That other authors have noted thermogenic and diuretic properties of caffeine, led Falk et al. (1989) to speculate that exercise under more stressful environmental conditions, or after higher doses of caffeine may hinder thermoregulation.

Food Iron Absorption Tea is a well known inhibitor of food iron absorption but studies have also shown that coffee significantly inhibits food iron absorption by as much as 83%. The inhibition of iron absorption is concentration-dependent (Morck et al., 1983). This has profound implications for endurance athletes who require sufficient iron for efficient oxygen transport around the body to feed working muscles. The time of caffeine ingestion has an important effect on iron absorption. When coffee and iron-rich food are taken together, or when coffee is ingested one hour after food, the degree of iron inhibition is similar, but there is no inhibition when coffee is taken one hour before food.

Toxicity Caffeine is not free of toxic effects. However, some effects are trivial in nature compared to the benefits derived from its use. In the modest doses usually used by athletes, it is unlikely to produce side effects more significant than some tremor and hyperactivity (phenomena not unknown to heavy coffee drinkers). However, the central excitatory effects of caffeine may cause an athlete to become oblivious to fatigue symptoms, even when they reach dangerously high levels. Fatigue is the expected consequence of sports competition and a part of the body's built-in biological warning system. Falls (1968) states:

> Drugs make men (sic) ignore danger signs which are a threat to man's (sic) physiological and psychological reserves.

As the consumption of caffeine increases, the effects on the body become more noticeable and severe. For a healthy adult, about two 150ml cups of brewed coffee (85 to 250mg) would decrease drowsiness, fatigue, and allow an easier flow of thought. For most people, this is the desired effect. However, a review of caffeine abuse research (Hughes et al., 1992) revealed that abstinence from caffeine induces a withdrawal syndrome of headache, fatigue, and drowsiness, that begins within 12 to 24 hours and lasts about one week. The syndrome can be severe and appears to be one reason for continued use of caffeine.

Caffeine also has psychotropic and addictive properties. It can produce a set of behavioural and physiological symptoms in people who consume high doses (500 to 1000mg daily), wh are caffeine-sensitive, and who are not accustomed to using caffeine. This is known as "caffeinism" or "caffeine intoxication" (DSMIII-R,1987), the symptoms of which are similar to anxiety neurosis (Shanahan & Hughes, 1986; Davis, 1990), and include restlessness, nervousness, excitement, insomnia, flushed face, diuresis, gastrointestinal disturbance, muscle twitching, rambling flow of thought and speech, tachycardia or cardiac arrhythmia, periods of inexhaustibility, and psychomotor agitation (Stavric, 1988). In sensitive individuals, caffeine has even caused convulsions and vomiting.

At doses greater than 1g per day, muscle twitching, rambling flow of thought and speech, cardiac arrhythmias, periods of inexhaustibility, and psychomotor agitation may also occur. Impaired memory, pronounced anxiety, and sensory disturbances including hallucinations are some of the results of ingesting massive doses of caffeine by humans under prolonged competitive stress (Stillner, 1978). The acute human fatal dose appears to be greater than 10g (or 170mg per kg body weight; Graham, 1978). Death may result from seizures and respiratory failure (DSMIII-R, 1987). The amount of coffee required to provide 5 to 10g caffeine has been reported variously as "40 cups of strong coffee over a short time" (Work, 1991) and about "75 cups of coffee... at one time" (Curatolo & Robertson, 1983). Other authors report a lower lethal dose (Watson, 1988), and even a small amount injected directly into the blood stream, can be fatal (Fisher & Jensen, 1990). However, deaths related to caffeine overdose are uncommon (Leonard et al., 1987), and only a few cases have been reported in the literature (Stavric, 1988).

CONCLUSION

The literature describing caffeine's effects on sport performance is vast and equivocal. Caffeine research faces almost every imaginable methodological difficulty including conflicting results in sound studies. In light of equivocal findings, the benefit of caffeine as an ergogenic aid is questionable and athletes should therefore be discouraged from using it. If a naive user ingests it there is no way of predicting what the results would be. Indeed, caffeine could harm a persons performance by causing irritability and nervousness. To encourage the use of caffeine to improve performance could be seen as an endorsement of the use of drugs, which would be both inappropriate and unethical. On the other hand caffeine use could be endorsed if it were seen as purely a nutritional aid, not unlike the practice of carbohydrate loading. Overall, it appears that moderate use of caffeine will not cause problems for most people. But athletes who are susceptible to its effects, such as those who respond with arrhythmias should avoid caffeine.

This project attempted to assess the effect of caffeine consumption on expectations of caffeine enhanced performance and caffeinism symptoms among New Zealand athletes. It was hypothesised that higher caffeine users would have greater performance-enhancement expectancies and would exhibit a greater number of caffeinism signs. This assumption was supported by the general body of literature describing the physiological effects of increased levels of caffeine consumption as well as two specific studies (Bradley & Petree, 1990; Page, 1987) which related caffeine consumption to expectancies of performance benefits. It was also hypothesized that athletes who had a strong athletic identity would be more likely to have used caffeine as an ergogenic aid. This assumption was made on the basis of a comment regarding the AIMS (Brewer et al., 1993). These authors suggested that a strong athletic identity may prompt individuals to engage in a sport behaviour that could jeopardize their physical health - this project tested this assumption using caffeine abuse as the behaviour in question.

This study found no relationship between normal caffeine intake and level of caffeinism. However a relationship was found regarding caffeine expectancies and the extent to which an athlete exhibits the symptoms of caffeinism. It was also found that subjects who have sport-performance expectancies are more likely to use caffeine as a performance enhancing

drug. Furthermore, athletes who took caffeine as an ergogenic drug were more likely to identify strongly with the athlete role.

Given a study with minimal methodological shortcomings, athletes' expectations of enhancing their performance through caffeine consumption may offer some promise as a predictor of the extent of caffeine consumption by athletes and their concomitant risk of caffeinism. This study contains some valuable information and could be treated as a pilot study for anyone wishing to continue with the research. Naturally the questionnaire must be refined, along with the method of subject selection and follow-up. Several other research suggestions are made which may clarify the links between caffeine and sport.

There are only a few documented cases of positive drug tests returned for caffeine in international competition. But this may be the tip of the iceberg. The banned level of caffeine is set so high by the IOC that caffeine is not recognised as a drug of 'abuse' at lower levels, despite the fact that athletes may be showing signs of caffeinism, a condition of clinical significance.

Additional scientific research to ascertain the extent of caffeine use in contemporary sport is needed. It is hoped that the results of this study will galvanise researchers to go beyond the limitations of the all-too-often reported anecdotal evidence. Instead, assessments of the relationships between the athlete's knowledge, attitudes, and drug-taking behaviours need to be undertaken in a systematic and scientific fashion. Only then can coaches, sport administrators, sport psychology consultants, and athletes work jointly to use caffeine safely and to inhibit the unethical and unhealthy use of the drug amongst the individuals who have traditionally been the healthiest models in our society, sports competitors. Nothing less than the integrity of the game is at stake. I leave the reader with a thought-provoking quote:

> *'In the old days, at competitions, people would cluster round the champions asking them about their exercise routines. Now they just ask 'What drugs are you on?'* -
> Body Builder Roger Wailker, 3rd Mr Britain, 1973 (Samuel, 1985).

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APPENDICES

Appendix 1

Beverages consumed at least once a week

		Fizzy	Decaf.	Coffee Substit.	Milo Cocoa	Coffee	Tea
TOTAL	(Age 15+)	42%*	19%	6%	32%	87%	93%
Males	1. 15-18	82	15	5	41	79	85
	2. 19-24	68	19	2	37	92	81
	3. 25-44	55	17	5	35	91	93
	4. 45-64	29	16	6	24	90	96
	5. 65+	17	21	5	24	87	99
	6. ALL	50	17	5	32	89	93
Females	1. 15-18	63	18	5	43	75	76
	2. 19-24	57	17	5	40	81	87
	3. 25-44	34	18	8	35	86	94
	4. 45-64	23	19	8	24	88	96
	5. 65+	16	27	7	23	85	98
	6. ALL	35	20	7	32	85	93

* Percentage of sample; Male respondents=2083, Female respondents=2603

Appendix 2

Frequency of Consumption of Beverages

		Fizzy	Decaf.	Coffee Substit.	Milo Cocoa	Coffee	Tea
TOTAL	(Age 15+)	3*	12	9	6	19	23
Males	1. 15-18	3	7	9	4	14	13
	2. 19-24	5	13	#	8	18	18
	3. 25-44	3	12	10	6	21	21
	4. 45-64	3	14	11	5	19	27
	5. 65+	3	7	5	6	13	28
	6. ALL	4	11	10	6	19	22
Females	1. 15-18	3	10	6	5	15	13
	2. 19-24	4	15	14	8	20	17
	3. 25-44	3	16	9	5	22	22
	4. 45-64	3	14	9	5	18	25
	5. 65+	2	10	6	5	13	29
	6. ALL	3	13	9	6	19	23

* Number of glasses/cups per week of those who consumed the beverage at least once per week.

Data from the Life in New Zealand Commission Report (Russel & Wilson, 1991).

Appendix 3

Sporting Involvement by New Zealanders over age 15	
Archery, Pistol/Rifle Shooting	3%
Athletics/Harriers	5%
Boxing/Wrestling	1%
Cycling	17%
Fencing	0%
Rink Sports	1%
Rowing	2%
Triathlon	no data
Body Building	no data
Power Lifting	no data

NB Participation required an involvement of greater than one month. Male respondents=1925, Female respondents=2448.

Data from the Life in New Zealand Commission Report (Russel & Wilson, 1991).

Appendix 4
Recommended Schedule of Testing for Banned Substances among the Various Sports

1. Associated Sports NZOCGA

SPORT	STIMULANTS (CAFFEINE)	NARCOTIC ANALGESICS	ANABOLIC STEROIDS	BETA-BLOCKERS	DIURETICS
Archery	Comp	OOC/Comp		OOC/Comp	
Track & Field	Comp	OOC/Comp	OOC/Comp	OOC/Comp	
Sprints and Jumps	Comp	OOC/Comp	OOC/Comp	Modern pentathlon	
Middle Distance	Comp	OOC/Comp	OOC/Comp		
Long Distance	Comp	OOC/Comp			
Boxing	Comp	OOC/Comp	OOC/Comp		
Cycling	Comp	OOC/Comp	OOC/Comp		
Fencing	Comp	OOC/Comp			
Ice Racing	Comp	OOC/Comp	OOC/Comp		
Rowing	Comp	OOC/Comp	OOC/Comp		
Shooting	Comp	OOC/Comp		OOC/Comp	
Weight Lifting	Comp	OOC/Comp	OOC/Comp		Comp
Olympic Wrestling	Comp	OOC/Comp	OOC/Comp		Comp

2. Non-Associated Sports

SPORT	STIMULANTS (CAFFEINE)	NARCOTIC ANALGESICS	ANABOLIC STEROIDS	BETA-BLOCKERS	DIURETICS
Triathlon	Comp	OOC/Comp			

Abbreviations: OOC = Out of competition Testing; Comp = In competition testing

Adapted from Hatherton et al. (1991).

Appendix 5
Letter to National Sport Organisations

Psychology Department
University of Canterbury
Ilam

1 July 1994

[National Sports Organisation]
[Address]
[City]

To the President

I am writing requesting the assistance of your association with some work I am undertaking to complete my MSc at the University of Canterbury. My thesis involves athletes' perceptions of caffeine, as well as the actual use of caffeine amongst new Zealand sports-people in different sporting codes.

My hopes are that National Sports Organisations will assist me in the data gathering stage of the process. I am proposing that I send your organisation 50 questionnaires for distribution by you to current registered athletes in your organisation. These athletes should represent all the grading levels from age 18 within the *[name of association].* All postage costs will be incurred by me, and your commitment, should you wish to take part in this, will be only to address the packages. However, if you could make a mailing list available to me, I can take over this responsibility also.

Each athlete will receive a consent form to be signed, and various questionnaires assessing their level of involvement in their sport, their consumption of caffeine, and their perceptions of caffeine. They will also have in their package a demographic questionnaire and a self-addressed pre-paid envelope in order to send the completed questionnaires back to me.

Assurance of complete confidentiality is given to every athlete partaking in this study, and they will also be given a summary of findings once the results have been collated. Your organisation will

also receive summary findings and sample questionnaires for your records.

Should you wish to be involved in this study, please drop me a line at the above address or fax number at your earliest possible convenience. I would be very grateful to have the assistance of the *[name of organisation]*, and trust that the results could be valuable to you.

Yours sincerely

Anita van Maanen
(Researcher)

Appendix 6
National Sports Organisations to whom Requests were Sent

Athletics New Zealand	replied, offered assistance
New Zealand Boxing Association Inc.	no reply
Cycling New Zealand	replied, offered assistance
Ice Racing Federation of New Zealand Inc.	replied, offered assistance
New Zealand Power Lifting Federation	no reply
New Zealand Rowing Association	replied, declined assistance
New Zealand Shooting Federation Inc.	no reply
Triathlon New Zealand	replied, offered assistance
New Zealand Olympic Wrestling Union Inc.	no reply
New Zealand Body Building	no reply
New Zealand Amateur Fencing Association	no reply
New Zealand Archery Association	no reply

Appendix 7
Final List of Organisations which Distributed Questionnaires

Athletics New Zealand
Canterbury Boxing Association
Cycling New Zealand
Ice Racing Federation of New Zealand Inc.
Canterbury Powerlifting Federation
Union Rowing Club (to rowers affiliated to NZARA in Canterbury)
Canterbury Small Bore Rifle Association
Triathlon New Zealand
New Zealand Olympic Wrestling Union Inc.
Representative of New Zealand Federation of Body Builders
Canterbury Fencing Association
Christchurch Archery Club

Appendix 8
Letter of Introduction to Athletes

Dear Athlete

I am writing requesting your assistance with some research into the use of caffeine as an ergogenic (performance-enhancing) agent and the consequences of caffeine use as athletes perceive them. Your name was randomly selected by the organisation to which you are affiliated, along with 549 other athletes in various sporting codes. The study, I hope, will be of interest, not only to you, but also to coaches, administrators, and athletes of other disciplines. Enclosed you will find questionnaires which are to be completed, and a consent form to be signed.

Please accept my assurance of complete confidentiality. The finished thesis will not include your name, and all records can be destroyed at your request. However, the aggregated results will be included in the final thesis.

The success of this research is dependent entirely upon your assistance in completing these questionnaires honestly. This involves some degree of commitment on your part but your organisation will subsequently receive beneficial information from your participation.

Please fill in these questionnaires and return them to me, along with the signed consent form as soon as possible. There is no postage required. If you have any questions, please do not hesitate to call me during office hours on (03) 364-2391 or after hours on (03) 327-7711. Thankyou for your assistance, and good training.

Kind regards,

Anita van Maanen (Researcher)

Appendix 9
Consent Form

UNIVERSITY OF CANTERBURY DEPARTMENT OF PSYCHOLOGY

CONSENT FORM

Reason for the project: To generate data about the use of caffeine by New Zealand athletes in various sports and to assess the beliefs these athletes have about the pharmacological, physiological, and psychological effects of caffeine administration.

Your tasks in this project: To complete and return the questionnaires issued to you.

Risks associated with participation: There are no risks associated with participation.

Confidentiality: All medical information and test results will be held in strictest confidence, and records destroyed at your request. Your organisation will be given a copy of results obtained from the study. This will include aggregated results from all subjects, and not the results from your individual participation.

Voluntary participation: Your permission to partake in this study is voluntary. You are free to deny consent or refrain from participation if you so desire. However, your assistance would be greatly appreciated.

Time required: The questionnaire pack should take between 15 and 30 minutes too complete.

Researcher: Anita L. van Maanen **Supervisor**: Dr. Rob Hughes

I agree to participate in the project described above, on the understanding that if at any time I wish to withdraw from the experiment I may, without prejudice, do so. All information collected will be confidential as will the identity of participants.

Name..... **Date** .....
Signature .....

Appendix 10

DEMOGRAPHIC QUESTIONNAIRE **DEMO-Q**

The purpose of this questionnaire is to obtain a general picture of your background. In scientific work, records are necessary, since they permit an accurate description of the study population. Please answer these routine questions in your own time. Please place the number of your choice in the box at the right of the page.

It is understandable that you might be concerned about what happens to the information about you, because much of this information is highly personal. All records are strictly confidential. No outsider is permitted to see this information without your permission.

Your gender

col. 5 ☐

1. Male
2. Female

Your age

☐

1. 18 - 25
2. 26 - 30
3. 31 - 35
4. 36 - 40
5. 41 - 45
6. 46 - 50

Gross personal income for 1994

☐

1. less than $10 000
2. $10 001 - $20 000
3. $20 001 - $30 000
4. $30 001 - $40 000
5. $40 001 - $50 000
6. more than $50 000

Total household income for 1994

☐ col. 8

1. less than $10 000
2. $10 001 - $20 000
3. $20 001 - $30 000
4. $30 001 - $40 000
5. $40 001 - $50 000
6. more than $50 000

Appendix 10 cont.

Relationship status col.9

1. single
2. married
3. de facto
4. widowed
5. divorced
6. separated

Number of children (born to you or under your care)

0. nil
1. one
2. two
3. three
4. four
5. over four

Ethnic origin

1. New Zealand European
2. New Zealand Maori
3. Polynesian
4. Asian
5. Other
   *specify .....

Highest qualification achieved

1. School Certificate
2. Sixth Form Certificate col. 12
3. University Entrance
4. Bursary 'B'
5. Bursary 'A'
6. Scholarship
7. University degree/diploma
   *specify .....
8. Other
   *specify .....

Appendix 11
Caffeinism Symptoms Questionnaire

CAFF-SX

To what extent do you purposely choose **non-caffeinated** beverages?

1. Always
2. Usually
3. Sometimes
4. Never

col. 14

Was the last drink/soft drink you consumed:

1. Caffeinated
2. Caffeine-free
3. Don't know

(If you responded ' Don't know', what was your last drink?_____

Does caffeine enhance performance in your sport?

1. Yes
2. No
3. Sometimes
4. Don't know

Please indicate how often you experience the following symptoms after drinking caffeine-containing beverages.

1. Never
2. Sometimes
3. Often

Restlessness

Nervousness

Insomnia (inability to sleep)

col. 19

Appendix 11 cont.

Please indicate how often you experience the following symptoms after drinking caffeine-containing beverages.

1. Never
2. Sometimes
3. Often

col. 20

Flushed face

Diuresis (frequent need to urinate)

Gastrointestinal disturbance

Muscle twitching

Impaired thought and/or speech

Pounding heart or palpitations

Periods of inexhaustibility

For office use only:

S = 1, SP
S = 2, SA

col. 27

Appendix 12

PERCEIVED CONSEQUENCES OF CAFFEINE EP-CAFF.1

This questionnaire is designed to assess <u>your</u> beliefs about the characteristics of caffeine and caffeinated beverages. Please indicate your answer by placing the number of the answer which best reflects <u>your</u> belief in the box at the right of the page.

1. **Yes**
2. **No**
3. **Unsure**

col. 29

Caffeinated beverages taste good..... ☐

Caffeine causes cancer..... ☐

Caffeine helps drunk people sober up..... ☐

Caffeine helps me feel better..... ☐

Caffeine helps me relax..... ☐

Caffeine makes people irritable..... ☐

Caffeine gives people high blood pressure..... ☐

Caffeine makes people urinate often..... ☐

Caffeine is bad for teeth..... ☐

Caffeine damages the kidneys and bladder..... ☐

Caffeine makes the heart beat faster..... ☐

col. 39

Appendix 12 cont.

1. Yes 2. No 3. Unsure

col. 40

Caffeine makes people gain weight..... ☐

Caffeine helps people think more clearly..... ☐

Caffeine makes people feel shaky and jittery..... ☐

Caffeinated beverages are refreshing..... ☐

Caffeine upsets the stomach..... ☐

Caffeine causes acne..... ☐

Caffeine makes people nervous and anxious..... ☐

Caffeine keeps people awake when they're tired..... ☐

Caffeine causes headaches..... ☐

Caffeine causes ulcers..... ☐

Caffeine is harmful to health..... ☐

Caffeine helps people perform better..... ☐

Caffeinated beverages are habit-forming..... ☐

Caffeine gives people more energy..... ☐

Caffeine helps people stay alert..... ☐

col. 54

Appendix 13

PERCEIVED CONSEQUENCES OF CAFFEINE EP-CAFF.2

This questionnaire is designed to assess <u>your</u> beliefs about the characteristics of caffeine as they relate to sport. Please indicate your answer by placing the <u>number</u> of the answer which best reflects <u>your</u> belief in the box at the right of the page.

1. Yes **2. No** **3. Unsure** col. 56

In my sport, caffeine enables one to work for longer before fatigue..... ☐

In my sport, caffeine increases fat utilisation..... ☐

In my sport, caffeine helps spare muscle glycogen stores..... ☐

In my sport, the supposed effects of caffeine are psychological..... ☐

The more caffeine consumed, the more performance improves..... ☐

Caffeine reduces tremors (for example, shaky hands)..... ☐

Caffeine can be of benefit in endurance sports..... ☐

Caffeine can be of benefit in sports requiring alertness..... ☐

Caffeine can be of benefit in sports requiring low anxiety levels..... ☐

Caffeine can be of benefit in sports requiring strength and power..... ☐

Caffeine can help athletes lose body fat..... ☐

Very high doses of caffeine can impair sports performance..... ☐

Caffeine masks fatigue/tiredness during sports training/competition..... ☐

col. 68

Appendix 14

SPORT SPECIFIC QUESTIONNAIRE **SPORT-Q**

This questionnaire is designed to get some information relevant to your involvement in sport. Please answer the questions honestly and to the best of your knowledge. Indicate your answers by placing the number of choice in the right hand box.

For office use only: col. 76

SPORT

What is the **main** reason you participate in sport?

1. Improve skills
2. Enjoy the competition
3. Maintain fitness
4. Wish to represent New Zealand
5. Recognition
6. Social contact

How many **years** have you been involved competitively in this sport?

1. Less than three years **2.** Between three and six years
3. More than six years

What is your **highest level** of participation?

1. Local community based competition
2. Regional competition
3. National competition
4. International competition

How many **hours** do train per week during in-season?

1. 0 - 7 **2.** 7 - 14 **3.** 14 - 21 **4.** More than 21

Do you consume caffeine for its performance enhancing effects?

1. Yes **2.** No **3.** Sometimes

If you answered **1** or **3**, how long have you been using caffeine as a performance enhancing drug?

1. Less than 1 year **2.** 1 - 2 years **3.** 2 - 3 years
4. 3 - 4 years **5.** 4 - 5 years **6.** More than 5 years

Have you ever intentionally used caffeine in competition to enhance performance knowing that you risk disqualification if urine levels of caffeine are high enough? col. 78

1. Yes **2.** No

Appendix 15
Athletic Identity Measurement Scale

AIMS

Please rate the way you feel in terms of the statements given below.

1. Strongly agree
2. Agree
3. Neither agree nor disagree
4. Disagree
5. Strongly disagree

col. 5

I consider myself an athlete. ☐

I have many goals related to sport. ☐

Most of my friends are athletes. ☐

Sport is the most important part of my life. ☐

I spend more time thinking about sport than anything else. ☐

I need to participate in sport to feel good about myself. ☐

Other people see me mainly as an athlete. ☐

I feel bad about myself when I do poorly in sport. ☐

Sport is the only important thing in my life. ☐

I would be very depressed if I were injured and could compete in sport. ☐

For office use only:

S = 1. AIMS-p 2. AIMS-a ☐

col. 15

Appendix 16

CAFFEINE INTAKE QUESTIONNAIRE **T-CAFF**

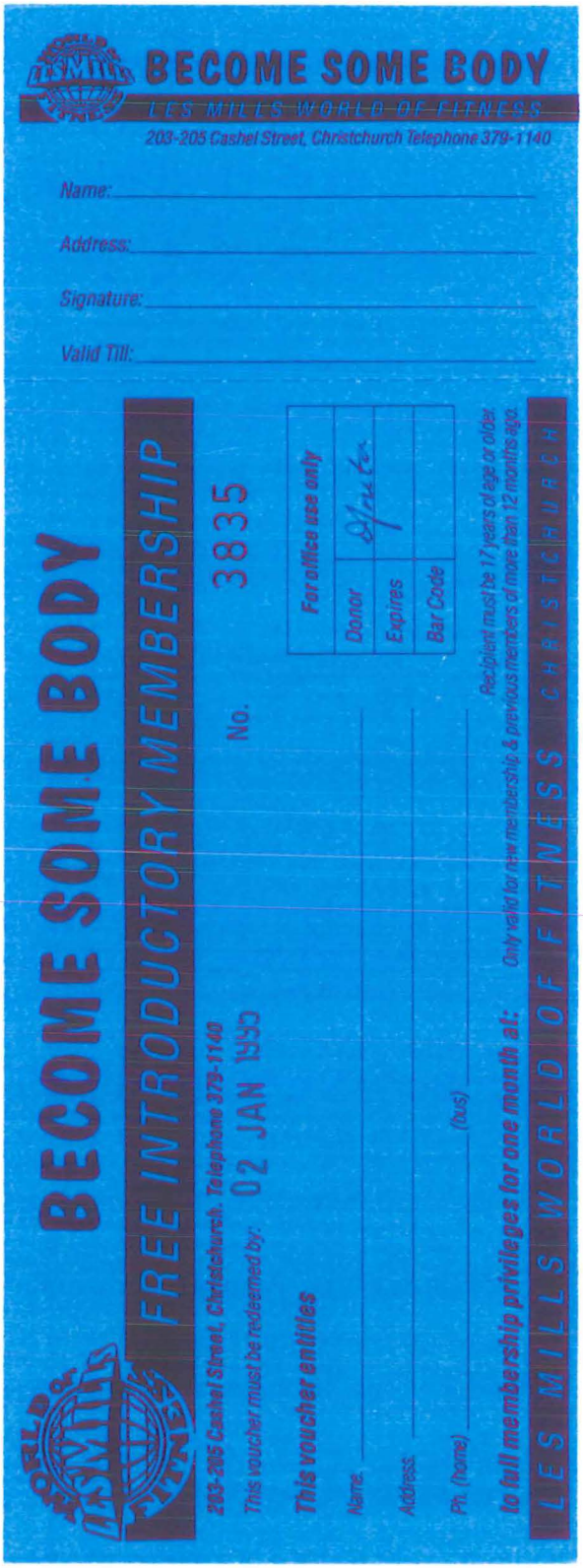
This questionnaire is designed to get some information about the amount of caffeine you consume. Please estimate your daily caffeine consumption by placing the quantity inside each bracket as applicable.

ITEM	***QUANTITY***
Coffee:	
Brewed, drip method	( ) cups
Brewed, percolator	( ) cups
Brewed, plunger	( ) cups
Instant	( ) cups
Decaffeinated, brewed	( ) cups
Decaffeinated, instant	( ) cups
Tea:	
Brewed	( ) cups
Instant	( ) cups
Cocoa	( ) heaped teaspoons
Milo/Bournvita	( ) heaped teaspoons
Milk chocolate	( ) grams (approx.)
Dark chocolate	( ) grams (approx.)
Cola drinks: (e.g. Pepsi, Coca-cola)	
Regular	( ) cans (333ml)
Diet	( ) cans (333ml)
Lucozade	( ) bottles (500 ml)
Prescription and Over-the-Counter Medications:	
Cafergot	( ) tablets
Fiorinal	( ) tablets
Soma Compound	( ) tablets
Darvon Compound	( ) tablets
Weight-Control Aids:	
please specify_____	( )
Alertness Tablets:	
please specify_____	( )
Analgesic/Pain relief:	
please specify_____	( )
Diuretics:	
please specify_____	( )
Cold/Allergy Remedies:	
please specify_____	( )

For office use only:

T-CAFF = Σ (CA) mg ( ).

Appendix 17
One-month Membership Gift



Appendix 18
Competition Entry Form

PLEASE ACCEPT THIS **FREE 1 MONTH MEMBERSHIP**
FOR YOU OR A FRIEND

AND GO IN THE DRAW FOR A **FREE**

6 MONTH MEMBERSHIP

AT LES MILLS WORLD OF FITNESS
IN CHRISTCHURCH

VALUED AT $395

JUST FILL IN THE DETAILS BELOW AND RETURN THIS SLIP, TOGETHER WITH YOUR COMPLETED QUESTIONNAIRE IN THE ENVELOPE PROVIDED.

NOTE: 1. Entries in the competition and signed consent forms are immediately separated from the questionnaire to keep your identity confidential. Furthermore, the green 3-digit identifier number on your questionnaire is not printed on either the consent form or the competition entry form in order to preserve your anonymity.

2. If you are not a Christchurch resident, you can transfer the membership into the name of a friend/relative who resides in Christchurch. Unfortunately the membership cannot be exchanged for cash or uplifted at another Les Mills World of Fitness branch.

3. I would like to thank Les Mills (ChCh) for the donation of memberships .

FREE 6 MONTH LES MILLS MEMBERSHIP DRAW

NAME: _____

PHONE: _____

Appendix 19
Distribution of Subjects According to AIMS Ratings

AIMS Item #	strongly agree	agree	neither agree nor disagree	disagree	strongly disagree
1. Cosiders self athlete	50.8%	32.4%	8.6%	6.5%	1.6%
2. Many sport related goals	44.3	37.3	6.5	11.9	0
3. Most friends are athletes	10.8	29.2	30.8	26.5	2.7
4. Sport most important	12.4	28.1	21.1	29.7	8.6
5. Thinks about sport often	16.8	14.1	21.6	31.9	15.7
6. Feel good when competing	19.5	35.1	19.5	21.1	4.9
7. People see me as athlete	14.6	32.4	24.9	23.2	4.3
8. Feel bad when lose at sport	22.7	41.1	16.8	16.2	3.2
9. Sport only important thing	2.7	5.9	16.2	39.5	35.7
10. Depressed if couldn't play	34.1	35.7	13.0	11.4	5.9

Appendix 20
Distribution of Subjects According to Caffeine Beliefs

ITEM # and Description	YES	NO	UNSURE
CNS Stimulating Effects			
13. Helps people think more clearly	21.6%	44.9%	33.5%
15. Is refreshing	65.9	25.9	8.1
19. Keeps people awake	76.2	10.8	13.0
23. Helps people perform better	38.4	27.6	34.1
25. Gives people more energy	35.7	36.8	27.6
26. Helps people stay alert	64.9	10.8	24.3
Other Perceived Positive Effects			
1. Caffeinated beverages taste good	71.4	15.1	13.5
3. Helps drunk people sober up	14.6	64.3	21.1
4. Helps me feel better	44.9	42.2	13.0
5. Helps me relax	25.9	57.8	16.2
Perceived Negative Effects			
2. Causes cancer	5.4	42.2	52.4
6. Makes people irritable	18.9	40.5	40.5
7. Gives people high blood pressure	22.2	27.0	50.8
8. Makes people urinate often	50.3	16.2	33.5
9. Is bad for teeth	24.3	34.1	41.6
10. Damages the kidney and bladder	16.2	25.9	57.8
11. Makes the heart beat faster	55.1	14.6	30.3
12. Makes people gain weight	4.3	63.2	32.4
14. Makes people feel shaky and jittery	30.3	41.1	28.6
16. Upsets the stomach	22.2	50.8	22.0
17. Causes acne	1.6	61.1	37.3
18. Makes people nervous and anxious	25.4	42.2	32.4
20. Causes headaches	29.7	40.5	29.7
21. Causes ulcers	9.7	42.2	48.1
22. Is harmful to health	33.0	34.1	33.0
24. Is habit-forming	76.8	11.9	11.4

Appendix 21
Distribution of Subjects According to Beliefs of Caffeine as a Performance Enhancing Drug in Sport

ITEM # and Description	*YES*	*NO*	*UNSURE*
Perceived Effects			
1. Enables one to work longer before fatigue in my sport	22.2	51.1	23.8
2. Increases fat utilisation in my sport	19.5	37.3	43.2
3. Helps spare muscle glycogen in my sport	11.4	37.8	50.8
4. Supposed effects are psychological in my sport	35.7	35.7	28.6
5. The more consumed, the more performance improves	13.5	68.6	17.8
6. Reduces tremor	11.9	54.1	34.1
7. Benefit in endurance sports	34.1	37.3	28.6
8. Benefit in sports requiring alertness	53.5	21.6	24.9
9. Benefit in sports requiring low anxiety levels	11.4	43.2	45.4
10. Benefit in sports requiring strength and power	22.2	48.6	29.22
11. Can help athletes lose body fat	18.4	41.1	40.5
12. Very high doses can impair sports performance	54.6	10.8	34.6
13. Masks fatigue/tiredness during sports	45.4	21.6	33.0

Appendix 22
Distribution of Athletes by Endorsement of Caffeinism Symptoms

CAFESX Item #	NEVER		SOMETIMES/OFTEN	
	FREQUENCY	%	FREQUENCY	%
1 *restlessness*	128	69.2	56	30.3
2 *nervouness*	161	87.0	23	12.4
3 *insomnia*	101	54.6	83	44.9
4 *flushed face*	169	91.4	14	7.6
5 *diureses*	73	39.5	111	60.0
6 *gastrointestinal disturbance*	142	76.8	42	22.7
7 *muscle twitching*	157	84.9	26	14.1
8 *impaired thought and/or speech*	164	88.6	20	10.8
9 *pounding heart*	145	78.4	39	21.1
10 *inexhaustibility*	142	76.8	41	22.2